Archives of Dermatology and Syphilology

VOLUME 24 NOVEMBER, 1931 NUMBER 5

THE AUTOHEMIC TREATMENT OF RHUS TOXICODENDRON DERMATITIS *

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In the summer of 1930 a patient came under my observation suffering from an extensive poison-ivy dermatitis, which had occurred during the three preceding summers. On his way home from the office he was in an automobile accident and sustained a fracture of the thigh, with extensive extravasation of blood at the site of injury. Within thirty-six hours the dermatitis disappeared. The question was, what had caused the serologic changes that led to the prompt disappearance of the dermatosis? The inference was, of course, that the extravasated blood had produced the phenomenon. That an individual's own whole blood outside its normal channels would obliterate this type of dermatitis seemed a reasonable hypothesis. This gave rise to the theory that in a case of poison-ivy dermatitis, the individual's own whole blood injected into the tissues would act as a curative agent.

Acting on this theory, the treatment was used in a series of cases, twenty of which are reported here. The technic of the treatment is to draw 10 cc. of blood from a convenient vein and immediately inject it deep into the gluteal muscles. Marked amelioration of signs and symptoms usually begins in the first twenty-four hours. If at the end of that time improvement is not evident, a second treatment is given; otherwise, the second treatment is given at the end of forty-eight hours. So far no patient observed has required further treatment. The pain and burning sensation rarely persist into the third day, and resolution is remarkably rapid.

Since the curative effect is apparent, the question of the prophylactic value of the treatment should be considered. In cases 13 and 15, both patients had experienced previous attacks with extensive involvement; both were treated within the first twenty-four hours after the onset of the attack reported, with prompt arrest of the process. This suggested a prophylactic effect.

Three farmers, who for a number of years suffered from ivy dermatitis starting early in the haying season, were each given one treatment on taking up their work in the hay fields. They all escaped the annual attack. No conclusion can be based on three cases, but they are suggestive. If the treatment does have an immunizing effect, extensive

* Submitted for publication, Aug. 12, 1931.

observation will be required to determine the duration of such an immunity.

As is well known, ivy dermatitis is frequently followed by an eczematous condition due to factors other than the ivy poisoning. This is doubtless true of those cases that persist beyond fourteen days. In some cases the eczema persists for months after the primary dermatitis. In none of the patients treated was there observed a subsequent eczema.

One attack is sometimes followed in a few days by a second, often due to contaminated wearing apparel. A patient came under observation a number of years ago, who suffered a severe attack of ivy dermatitis lasting for ten days. When he was able to be out, two

Results of Treatment in Twenty Cases

Case	Duration of Attack Before Treatment, Days	Duration of Attack After Treatment, Days	Number of Previous Attacks	Average Duration of Previous Attacks, Days
1	2	2	4	10
2	3	2	3	14
3	2	1	7	14
4	5	3	2	20
5	2	2	None	..
6	3	1	2	10
7	2	2	15	15
8	7	2	None	..
9	2	2	3	14
10	5	2	5	15
11	2	3	4	10
12	1	3	None	..
13	1	2	6	7
14	2	2	None	..
15	1	1	4	10
16	4	3	3	20
17	2	1	1	7
18	2	3	2	15
19	1	1	None	..
20	3	4	2	12

weeks after the onset, he wore the same golf hose that he had worn when first attacked. He suffered a severe recurrence, beginning on his legs and hands. The only possible source of contamination was the unlaundered hose. So far, no recurrence has taken place in the series of cases in which treatment was given.

COMMENT AND CONCLUSIONS

In a series of cases of Rhus toxicodendron dermatitis, the patient's own whole blood was injected into the muscular tissues. This treatment was suggested by the rapid disappearance of this type of dermatitis following a trauma with extravasation and ecchymosis.

The effect of the treatment has justified the conclusion that the patient's own whole blood is a rapidly acting specific, and probably of prophylactic value.

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JUGULAR COMPRESSION

AN ADJUNCT IN THE TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM *

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The relative ineffectiveness of most remedies for diseases of the central nervous system is a subject of much interest and importance. That a great number of chemical and biologic substances do not penetrate into nerve tissue and the spinal fluid in appreciable concentration is a well known fact. Under normal conditions there is a competent barrier between the nervous system and many of the ingredients of the blood within the cranial and the spinal vessels. This has been called the hemato-encephalic barrier. It is illustrated by the absence of staining of nerve tissue and spinal fluid in cases of jaundice. The numerous recommendations, both as to remedy and as to method, concerning the treatment for neurosyphilis confirm these statements.

Some of the suggested explanations of this barrier are: (1) the atypical vascular distribution and the absence of lymphatics, (2) the inelasticity of the covering of the brain and the spinal cord, (3) the selective secretory (or dialyzing) action of the choroid plexus and (4) the peculiar chemical composition of the nerve cells and fibers.

The elimination of these difficulties could be achieved: (1) by the discovery of a remedy with the properties of affinity for nerve tissue, curative of syphilis and with low toxicity, or (2) by the finding of a way of increasing the effectiveness of the remedies already known and used in the treatment for neurosyphilis. Stated differently, the ratio between the maximum tolerated dose and the minimum amount therapeutically effective for the central nervous system should be improved.

The general subject of tissue or organ selective localization presents fascinating and pertinent problems. Ducrat and Gautrelet [1] reported a paralysis of the selective secretory action of the choroid plexus by the

* Submitted for publication, Feb. 8, 1931.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

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1. Ducrat and Gautrelet: Compt. rend. Soc. de biol. **1**:161, 1905.

intracarotid injection of methyl violet. Following this suggestion, we [2] found that the intravenous injection of methyl violet did not increase the permeability of the choroid plexus to arsphenamine in dogs.

As it was observed that jugular compression promptly increased the intracranial pressure and the rate of flow of spinal fluid, it was thought that possibly this mechanical procedure might increase the effectiveness of the antisyphilitic remedies for syphilis of the central nervous system. The passive hyperemia and venous stasis produced could conceivably cause a pouring forth of arsphenamized antisyphilitic serum into the ventricles, the subdural spaces and the parenchyma. The slowing of the blood stream with the stretching of the walls of the vessels might allow the curative agents, whether specific chemicals or immune substances, to reach the pathologic process. It was thought that jugular compression could be used without harm in selected cases. Some of the conditions in which the procedure is contraindicated or should be used cautiously are hemorrhagic diseases, rupture of an artery, neoplasms, glaucoma, separation of the retina, hydrocephalus and optic edema.

DaCosta,[3] in discussing the hyperemic treatment for inflammation advocated by Bier, stated that it hastens cure in many chronic inflammations. The effect is thought to be due to an increase in leukocytes and bactericidal serum, an increased permeability of the vascular structures and an improved nutrition. If this is true, one could conclude that jugular compression would increase the utilization of the arsphenamized spirocheticidal blood serum in the central nervous system and thereby hasten the resolution of the abnormal condition.

Weed and his co-workers [4] were unable to produce experimental meningitis in cats by intravenous injections of small doses of bacteria. When the same dosage was followed by two minutes of bilateral jugular compression, meningitis developed in 50 per cent of the animals. They stated that in their small animals it was impossible to be sure that the compression was properly applied, and expressed the opinion that the percentage of positive experiments would be increased by effective jugular compression. Flexner and Amoss [5] had previously found that a sterile meningitis facilitated the passage of poliomyelitis virus from the blood to the central nervous system. Kolmer [6] expressed the opinion

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2. Smith, D. C., and Waddell, J. A.: Am. J. Syph. **8**:310 (April) 1924.

3. DaCosta: Modern Surgery, Philadelphia, W. B. Saunders Company, 1929.

4. Weed; Wegeforth; Ayer, and Felton: Rockefeller Institute for Medical Research, Monograph Series no. 12, 1920.

5. Flexner, S., and Amoss, H. L.: J. Exper. Med. **25**:525, 1917.

6. Kolmer, J. A.: The Human Cerebrospinal Fluid, New York, Paul B. Hoeber, Inc., 1926, p. 79.

that in acute and passive congestion of the meninges antibodies from the plasma may be passed into the spinal fluid. Krogh,[7] in discussing the parallelism between capillary dilatation and increased permeability, said, "I find no case in which a considerable dilatation has taken place without being accompanied by an increase in permeability." He[8] also demonstrated that capillary pressure bears a direct relationship to venous pressure.

There is, then, both theoretical and experimental evidence to support the idea that jugular compression is a useful adjunct in treatment in selected cases of neurosyphilis. It might be beneficial in the treatment for some other diseases of the central nervous system and of the tissues about the head. Furthermore, it might be a useful addition to certain diagnostic measures as, for example, the Walter bromide coefficient. Additional evidence of the value of jugular compression in neurosyphilis can be obtained by: (1) the determination of the arsenic content of the spinal fluid following its use in conjunction with the injection of arsphenamine, (2) the determination of the arsenic content of the brain and spinal cord of animals following the injection of arsphenamine and jugular compression, (3) postmortem studies and (4) the clinical response. The present presentation deals only with the first of these considerations.

EXPERIMENTAL DATA

As there is little information about the details of the duration of the compression or the time of its application in relation to the injection of the arsenical, it was necessary to select these arbitrarily. The maximum concentration of arsenic in the blood stream occurs immediately following the injection.[9] A period of thirty minutes of compression following the injection of arsphenamine was used. It may be that compression preceding and following the administration of the drug might be more effective. This idea is based on the suggestion of Weed and his co-workers[4] that venous congestion and increased intracranial pressure force a more rapid absorption of spinal fluid, thus allowing an increased volume of blood within the fixed cavity.

The accompanying illustration shows the appliance used in compressing the jugular veins. There are two leather pads attached to a woven strap of sufficient length to encircle the neck and with an adjustable buckle. Each pad has a metal button on its outer flat surface which fits

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7. Krogh, A.: The Anatomy and Physiology of Capillaries, New Haven, Conn., Yale University Press, 1929, p. 335.

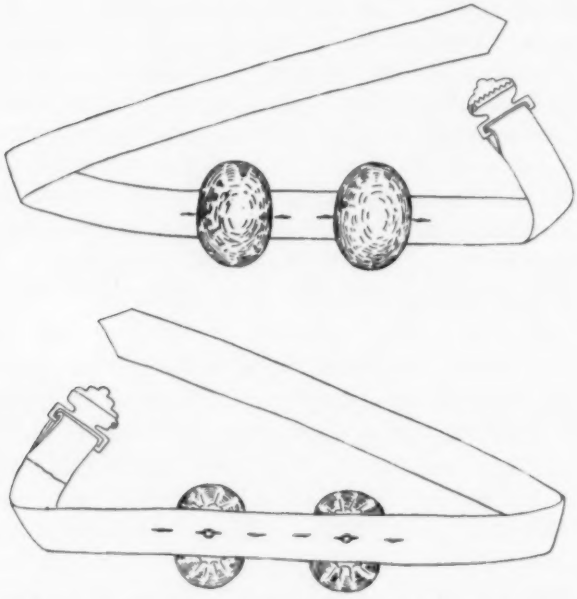
8. Krogh (footnote 7, p. 297).

9. Fordyce, J. A.; Rosen, I., and Myers, C. W.: Am. J. Syph. **8**:193 (April) 1924.

into the buttonhole of the strap. Engorgement of the veins of the face and the presence of pulsation in the temporal arteries are the indexes to the proper adjustment of the appliance. This causes the patient no, or very slight, discomfort.

The Gutzeit method of quantitative determination of arsenic was used. The results in eighteen control cases and in twenty-two cases of jugular compression are enumerated in tables 1 and 2.

The average arsenic content of the spinal fluid in the controls was 0.002 mg. per five cubic centimeters. Two of the eighteen control fluids



Appliance for jugular compression (about one-third the actual size).

were negative for arsenic. The average arsenic content of the spinal fluid in the cases of jugular compression was 0.0031 mg. per five cubic centimeters. There was an increase on the average of 55 per cent. All of the specimens of spinal fluid removed following jugular compression gave positive reactions for arsenic.

We have no explanation of the marked variation in the arsenic content of both control and test specimens. Efforts to correlate the irregularities in the integrity of the hemato-encephalic barrier with the clinical observations were unsuccessful. However, the general level and average of arsenic content of spinal fluid are raised by jugular compression.

The evidence presented by this small series is not conclusive; it is merely offered as suggestive. This report is, accordingly, only intro-

TABLE 1.—*Tests—Jugular Compression for Thirty Minutes Following the Intravenous Injection of Arsphenamine, Then Withdrawal of Spinal Fluid*

No.	Case	Intravenous Injection of Arsphenamine, Gm.	Status of Treatment*	Diagnosis	Mg. of Arsenic in 5 Cc. of Spinal Fluid
1	G. W.	0.2	2d of 1st course	Syphilis, history and serology	0.007
2	J. R.	0.3	2d of 1st course	Syphilis, secondary	0.006
3	J. C.	0.3	1st of 3d course	Syphilis, central nervous system; optic atrophy	0.005
4	F. H. H.	0.2	8th of 1st course	Syphilis, tabes dorsalis	0.005
5	W. W. W.	0.3	7th of 2d course	Syphilis, central nervous system, meningovascular	0.005
6	O. L.	0.2	1st of 2d course	Syphilis, serology	0.005
7	F. R.	0.2	2d of 1st course	Syphilis, secondary	0.004
8	H. B. S.	0.3	1st of 5th course	Syphilis, tabes dorsalis	0.004
9	A. F.	0.3	3d of 1st course	Syphilis, secondary	0.004
10	E. K.	0.3	2d of 1st course	Syphilis, secondary	0.003
11	J. S.	0.3	2d of 1st course	Syphilis, chancre	0.003
12	J. P. S.	0.3	3d of 1st course	Syphilis, history and serology	0.003
13	J. W.	0.3	3d of 1st course	Syphilis, central nervous system, meningovascular	0.0025
14	W. W. W.	0.3	1st of 2d course	Syphilis, central nervous system, meningovascular	0.002
15	D. G. B.	0.2	1st of 2d course	Syphilis, tabes dorsalis	0.002
16	R. Y.	0.4	2d of 1st course	Syphilis, secondary	0.002
17	M. C. A.	0.4	9th of 1st course	Syphilis, dementia paralytica	0.001
18	E. K.	0.4	3d of 1st course	Syphilis, secondary	0.001
19	J. W.	0.2	1st of 1st course	Syphilis, central nervous system, meningovascular	0.001
20	J. T. J.	0.4	1st of 4th course	Syphilis, dementia paralytica	0.001
21	N. W.	0.3	1st of 1st course	Syphilis, congenital, eighth nerve	0.001
22	F. W. A.	0.3	1st of 5th course	Syphilis, tabes dorsalis	0.001
	Average mg. of arsenic in 5 cc. of spinal fluid				0.0031

* A course of arsenical treatment usually consists of eight intravenous injections of arsphenamine at weekly intervals. The average dosage is 0.1 Gm. per forty pounds of body weight.

TABLE 2.—*Controls—Spinal Fluid Withdrawn Thirty Minutes After Intravenous Injection of Arsphenamine*

No.	Case	Intravenous Injection of Arsphenamine, Gm.	Status of Treatments	Diagnosis	Mg. of Arsenic in 5 Cc. of Spinal Fluid
1	M. D.	0.3	2d of 1st course	Syphilis, pharynx, gumma	0.006
2	J. F. R.	0.3	1st of 4th course	Syphilis, secondary	0.004
3	L. W.	0.3	1st of 7th course	Syphilis, tabes dorsalis	0.004
4	M. W.	0.3	1st of 4th course	Syphilis, secondary	0.004
5	L. T.	0.3	3d of 1st course	Syphilis, osteoperiostitis	0.003
6	H. E.	0.3	1st of 5th course	Syphilis, dementia paralytica	0.0025
7	C. W.	0.2	1st of 1st course	Syphilis, history and serology	0.002
8	B. H. K.	0.2	1st of 6th course	Syphilis, dementia paralytica	0.002
9	T. W.	0.3	1st of 4th course	Syphilis, central nervous system, meningovascular	0.002
10	W. V.	0.3	1st of 3d course	Syphilis, history and serology	0.001
11	W. F. P.	0.3	4th of 1st course	Syphilis, serology	0.001
12	A. R.	0.3	3d of 1st course	Syphilis, taboparesis	0.001
13	L. M.	0.2	1st of 2d course	Syphilis, spastic paralysis	0.001
14	G. S. T.	0.3	1st of 1st course	Syphilis, history and serology	0.0005
15	W. W.	0.4	2d of 1st course	Syphilis, secondary; central nervous system; optic neuritis	0.0005
16	R. S.	0.2	2d of 1st course	Syphilis, history and serology	0.0005
17	E. B. J.	0.3	8th of 1st course	Syphilis, chancre	0.000
18	I. K.	0.3	1st of 2d course	Syphilis, serology	0.000
	Average mg. of arsenic in 5 cc. of spinal fluid				0.0020

ductory. There are many details to be determined by further experimentation. The most effective time and duration are not settled. Intermittent compression may be an improvement. It may be found that jugular compression simultaneous with the secondary rises of arsenic content in the blood is an additional gain. The final answer desired, of course, is whether or not this procedure of jugular compression is an aid in the treatment for neurosyphilis or for other pathologic conditions.

SUMMARY

1. A small series of cases showed an increase of 55 per cent in the arsenic content of the spinal fluid following jugular compression as compared with controls. All of the test cases showed the presence of arsenic.

2. Theoretical and experimental data support the idea that jugular compression is a useful adjunct in treatment in selected cases of neurosyphilis.

ABSTRACT OF DISCUSSION

DR. HENRY E. MICHELSON, Minneapolis: I did not appreciate the full significance of Dr. Smith's contribution until I read the paper Professor Kolle presented before the Frankfort Dermatological Society recently. He has shown that with sufficient dosage he could entirely eliminate spirochetes from the brain of inoculated mice, while repeated small doses did not bring about a disappearance, showing that the action of the drug is direct and that there is a direct relationship between dosage and the extinction of the spirochete.

DR. EARL D. OSBORNE, Buffalo: I have been much interested in this subject in connection with my work on arsenic in the central nervous system. The idea of increasing the blood supply to the parenchyma of the brain by causing a vasodilatation seems to me an excellent one. It must be remembered, also, that fever therapy in one form or another produces a marked vasodilatation with a fall in the blood pressure. Instead of jugular compression, fever therapy might be substituted with even more beneficial results. It is an idea that should be worked out experimentally. We have had this in mind in relation to our work on arsenic in the central nervous system of rabbits. I also want to point out that the chemical determination of arsenic in the spinal fluid is not necessarily an accurate index of the amount of arsenic in the parenchyma of the brain itself. Just because arsenic in one form or other passes into the spinal fluid is no criterion that any of it has gained entrance to the parenchyma of the brain or spinal cord. As pointed out in our paper, we intend later to do microchemical studies on the brain and spinal cord, following the administration of arsenicals during fever therapy.

DR. D. C. SMITH, University, Va.: I would like to thank Dr. Michelson for his reference.

I did not mean to say that the estimation of the arsenic content of the spinal fluid is necessarily a proof of the curative value of jugular compression; it is only an indication. There could be a marked increase in arsenic in the parenchyma of the central nervous system without an increase in the spinal fluid. The use of the microchemical method of finding arsenic in tissue as employed by Dr. Osham would seem to be an improvement over examination of the spinal fluid.

We plan to carry forward these investigations.

THE RELATIONSHIP OF THE SEROLOGY OF THE BLOOD TO THAT OF THE SPINAL FLUID IN CONGENITAL SYPHILIS*

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The voluminous writings having to do with syphilis, inherited or acquired, fall far short of solving all of the problems of this protean disease. One is impressed by the complaints of clinicians concerning the multiplicity of tests. The foundation for the complaint would seem to be in direct proportion to the ability of the physician to interpret the tests. In this report effort has been made to determine the relationship between the serology of the blood and that of the spinal fluid in congenital syphilis, and, in addition, to determine if a child infected by a mother with latent syphilis is in a different position from the child infected by a mother with early syphilis.

The opportunity for this study was afforded by the James Whitcomb Riley Hospital for Children, a member of the Indiana University Hospital group. The patients in this hospital are all residents of the state of Indiana, and are received uniformly from all parts of the state, under a special statute enacted for the operation of this hospital. All children, from birth up to 16 years of age, with any illness, who are entitled to charity as determined by the judges of the circuit courts, are received as rapidly as the facilities of the hospital will permit; therefore, this study may be said to represent a fair cross-section of one of the middle western states as to the prevalence of congenital syphilis in its population.

In the present group of 103 cases of congenital syphilis, no effort was made to determine the serology of the spinal fluid until a diagnosis of syphilis had been made, either by anamnesis including serology of the blood or by clinical observations, or both.

If one is to gain a comprehensive grasp of the disease so contracted, many things, such as the stage of the disease existing in the mother at the time it is transmitted to the child, the developmental stage of the unborn child at the time the disease is contracted, the influence of the quiescent state of the child on the dissemination of the infection over the body and the effect of syphilotoxemia in early, as well as in latent, syphilis on the mother, must be given careful consideration.

For obvious reasons, little or nothing is known of the so-called primary stage of congenital syphilis. Therefore, with the exception of the infections that occur intra partem, I shall not give further consideration to this stage of the disease, other than to call attention to the early

* Submitted for publication, Feb. 15, 1931.
* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association in June, 1930, at Toronto, Canada.

development of the visceral portion of the body as compared with the integumentary tissues and the consequent result of the extensive invasion of these visceral tissues as a result of obtaining blood direct from the placenta.

It would seem that there are some who believe that the infection reaches the child by way of the ovum or the sperm, or both, and yet others who believe that the infection, for the most part at least, reaches the child by way of the mother's blood stream through the placenta. I think that the latter view is most often correct.

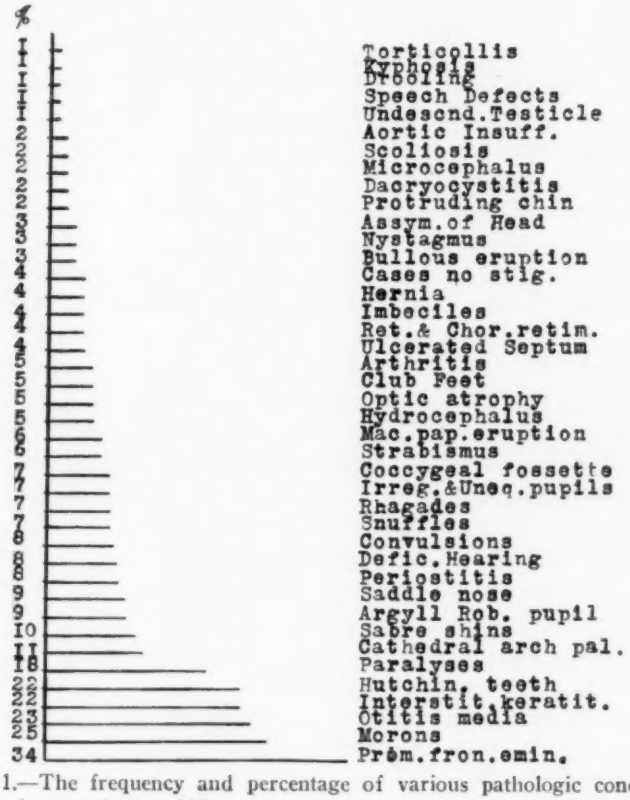

Chart 1.—The frequency and percentage of various pathologic conditions in 103 cases of congenital syphilis.

While some entertain the view that the child is most frequently infected by the mother when she is in the early stage of syphilis, it would seem that this would be the most plausible explanation for the termination of so many pregnancies as the result of this disease. Conversely, the infection of the child by the mother with latent syphilis accounts for the large number of children who have survived the infection in utero, only to reveal it later in life.

In the consideration of this subject, I shall take no account of the potent influences of therapy. If one is to visualize the situation, one

must have a clear conception of what constitutes early and what constitutes latent syphilis. By early syphilis, I mean the disease existing in the host up to the time that the immunologic processes have developed to their highest point; and by latent syphilis, I mean syphilis in the host from the time the immunologic processes have developed to their highest point throughout the remainder of life. That early syphilis may exist over a variable period of time, owing to therapy or failure on the part of the host to develop his natural immunity for reasons that are not well understood, there can be no doubt.

Infection by the mother while in the early stage of syphilis seems to account for the high degree of mortality observed, and the degree may

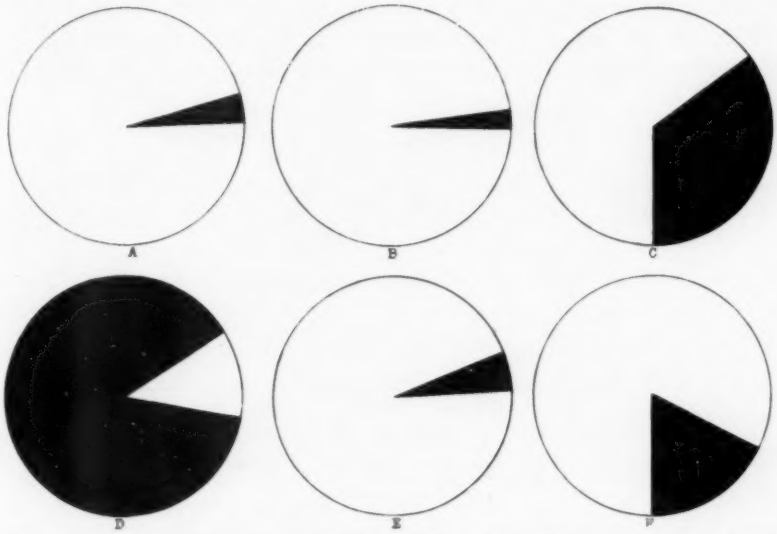

Chart 2.—*A*, frequency of congenital syphilis. The white area represents 7,000 cases admitted to Riley Hospital for all causes; the black area, 180 cases of congenital syphilis, or 2.5 per cent. *B*, frequency of cases with positive Wassermann reactions of the spinal fluid and negative reactions of the blood (dark area, 2.5 per cent). *C*, frequency of cases with a positive Wassermann reaction of the spinal fluid (dark area, 28 per cent) in the series of 103 cases. *D*, comparison of number of patients with early syphilis (light area, 15 per cent) with those with latent syphilis (dark area, 85 per cent). *E*, frequency of cases with globulin only in the spinal fluid and a positive Wassermann reaction of the blood. *F*, frequency of cases with colloidal gold curves (dark area, 18 per cent) in the series of 103 cases.

be said to be in direct proportion to the stage of pregnancy in which infection occurred; thus, when early syphilis occurred in the first few months the pregnancy would surely be terminated, either by the destruction of the child or by toxic products that would terminate the pregnancy before the child was viable. Moreover, a child in utero encountering

such an infection late in intra-uterine life may survive the infection and be born with early syphilis, possibly being clinically free from symptoms at birth, only to present generalized manifestations of syphilis (which bespeaks its early stage) as soon as it encounters the activating influence of irritation incidental to its change in environment.

In early syphilis in the mother, one visualizes the blood stream as carrying enormous numbers of spirochetes, and when the infection has once passed over to the placenta, the child's blood stream is immediately invaded by the same enormous number of organisms. However, in the mother with latent syphilis, this picture may be materially changed. In the first place, as pregnancy is a physiologic process, there is nothing about it per se to activate the syphilis; therefore, the blood stream may remain free from organisms throughout the pregnancy, and thus the child may escape; on the other hand, the mother may encounter some activating influence in the way of trauma, infection, etc., which may

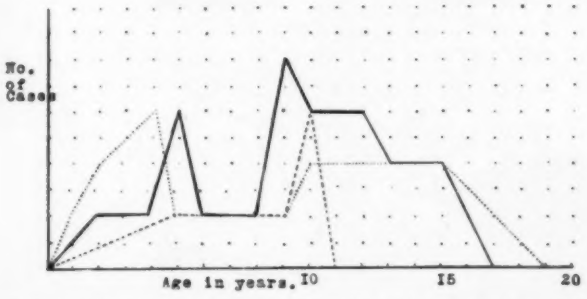

Chart 3.—Age incidence of interstitial keratitis (solid line), paralyses (dotted line) and periostitis (broken line) in 103 cases of congenital syphilis.

activate the latent disease. If so, the child may be infected, but the blood stream will not carry nearly as large a number of organisms as it would in early syphilis; therefore, the child's infection will not be so overwhelming. Furthermore, the child will benefit in its power of resistance by living in, and receiving its nourishment from, an immunized source. This would seem to explain some of the well recognized stigmas observed in congenital syphilis. For example, bosses, saddle nose and saber shin are dystrophies resulting from disturbances at the seventh or eighth week of intra-uterine life of the erupting centers of the frontal bones, the nasal bones and the tibias, whereas early decay of the first molar teeth (said by some to be pathognomonic of inherited syphilis) suggests that the child was born with early syphilis, for these centers of teeth erupt at birth and receive their disturbing influences, which produce the dystrophy as a result of the activating influence of the irritation attending the changed environment of the child at birth.

resulting in nasal, oral and genital irritations, and consequently symptoms, the generalization of which speaks for early syphilis.

Ordinarily, one thinks of inherited syphilis as invasion of the body of the unborn child by syphilitic organisms. I do not believe that this is necessarily true so far as serology is concerned. It would seem, and I believe that it does occur, that an early syphilotoxemia may be so severe that a fetus may perish without its body having been invaded by the organism, and that a latent syphilotoxemia may be the cause of a terminated pregnancy. Again, a latent syphilotoxemia may explain by its low grade persistence a class of infants born with an undernourished, shriveled, old, weazened appearance, with so-called inaptitude for life. In either case the infants' serums at birth may give positive reactions. This is equivalent to saying that positive serums may be the result of a syphilotoxemia or that the positive serums of the mother may pass to the child without the organism having done so. I believe this to be possible.

COMMENT

Of the 7,000 patients received at the James Whitcomb Riley Hospital for Children, 180 were found to be suffering from syphilis; this would show 2.5 per cent of infection with this disease in the population of Indiana. This percentage of infection is comparable with that of the army, which includes only one sex.

The discrepancy between the 103 cases concerned in this report and the 180 cases received in the hospital is due to the fact that in 77 cases serologic tests of the spinal fluid were not made. The tests were not thought justifiable (1) because of fear of damage to the nerve centers in some of the cases of early syphilis; (2) because of the gravity of the patient's physical condition, or (3) because residence in the hospital was not sufficiently long for the work to be done.

CONCLUSIONS

The child in utero encountering early syphilis in the mother during its early or middle uterine life most surely faces destruction if therapy is not instituted, whereas the child in utero encountering latent syphilis in the mother has a better chance to survive. It may suffer greatly from a prolonged syphilotoxemia, and represents the large number of patients showing dystrophic stigmas.

The Wassermann test and color reactions are of distinct service in inherited syphilis, whereas tests for albumin and cell counts are of service only after other infections have been ruled out.

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ABSTRACT OF DISCUSSION

Dr. Charles C. Dennie, Kansas City, Mo.: The observations on the cerebrospinal fluid in cases of congenital syphilis in infants are never so definite as those in cases in adults. Especially misleading, in view of the other observations, is the colloidal gold curve. A good many juvenile paralytic and tabetic patients may give positive evidence in all other tests when the gold curve is negative. In juvenile dementia paralytica and tabes the observations on the cerebrospinal fluid are not nearly so definite as when the diseases occur in adults. The percentage of positive cerebrospinal fluids, in my experience, decreases from birth to the age of 15. The same is true of the patients who have marked cutaneous syphilis in the early years. We know that if these diseases occur in the early years they will appear before the age of 5, and we find the positive evidence in the cerebrospinal fluid in a high percentage of these cases, somewhere over 50 per cent; as the age of 15 is reached, the positive observations in the cerebrospinal fluid fall to from 15 to 20 per cent. There must be, then, in the interval, a spontaneous disappearance of the signs of involvement of the cerebrospinal fluid without treatment.

Perhaps the most interesting group is that in which the children's mothers have received treatment before the child is born. The observation that if treated the mother will have a living child, whereas if not the child will be stillborn, is a very dangerous statement. It is true that a treated mother will give birth to living children, without a positive Wassermann reaction, but if you examine the cerebrospinal fluid you will be surprised to find that a large percentage of these fluids will be positive, and at the age of 5 years the Wassermann reaction will begin to drop back and signs of cerebrospinal syphilis will develop.

I think that the mothers should be treated most thoroughly, and that the treatment should be given before the fourth month of pregnancy. If in the natural course of events the child is going to bear the stigmas of syphilis, it will bear them anyway, in spite of treatment after the fourth month. I believe that insufficient treatment stimulates the production of cerebrospinal syphilis.

Dr. Frank W. Cregor, Indianapolis: Little is to be found in the literature concerning spinal and blood serology in congenital syphilis; therefore, I did not expect much discussion along that line.

However, my attention has recently been called to the importance of the effect of a syphilotoxemia, and I had hoped when the paper stated that a "syphilotoxemia might be the cause of a positive serology" it would elicit more discussion.

THE PRESENCE OF UNEVEN QUANTITIES OF BISMUTH IN OILY BULK SUSPENSION OF BISMUTH SALTS*

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Our attention was first called to the possibility of unequal amounts of bismuth in measured doses of oily bulk bismuth suspension when working with tartroquiniobine. This is a suspension of the iodobismuthate of quinine and of sodium potassium bismuth tartrate in camphorated oil and has a metallic bismuth content of 6 cg. per two cubic centimeters. Because of the time required when doses are put up in separate ampules, as well as because of the expense, we used this preparation in 25 cc. amounts put up in wide-mouthed shallow bottles. The bottles were thoroughly shaken before the dose was removed; moreover, the suspension contained several glass beads to assist in more thorough mixing. At the bottom of the bottle we were occasionally surprised to find a heavy, thick, reddish deposit. This disturbing factor led us to examine several other bulk products that we were using, the potassium bismuth tartrate suspension in oil, the bismuth salicylate suspension in oil and the bismuth salicylate cream suspension in oil.

In order to get the facts in the situation, samples of the different salts were taken from bottles in our office and in the clinics at the Lakeside and Cleveland City Hospitals. Each time before the samples were removed, the bottles were thoroughly shaken, as nearly as possible for a routine length of time. Samples were removed at random at different levels from the receptacles, and, moreover, samples were obtained at different times of the day, after various quantities had been taken out of the bottles.

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* Submitted for publication, Feb. 8, 1931.

* Drs. J. V. Ambler, Henry DeWolf and R. L. Howard helped in the collection of specimens.

* This investigation has been supported by a grant from the Committee on Research in Syphilis, Inc.

* From the Department of Dermatology and Syphilology and the Department of Pharmacology and Therapeutics, Western Reserve Medical School and the Cleveland City and Lakeside Hospitals.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

According to the manufacturers, our 20 per cent suspension of the potassium bismuth tartrate in oil is supposed to contain 0.128 Gm. of metallic bismuth per cubic centimeter; our bismuth salicylate cream suspension, 0.064 Gm. per cubic centimeter, and the bismuth salicylate preparation in oil, 0.0717 Gm. per cubic centimeter. With tartroquiniobine, analysis in the American Medical Association laboratory shows 0.06 Gm. of metallic bismuth per two cubic centimeters.

Our analyses were made according to the Warren method used by the Council on Pharmacy and Chemistry of the American Medical Association for the salicylate and for the tartrate. It consists roughly in the

TABLE 1.—*Analysis of Potassium Bismuth Tartrate*

Level of Liquid in Bottle	Bismuth, Gm. per Cc.	Average of Samples From Bottle	Variation From Average	Variation, per Cent	Level of Least Bismuth Recovered	Level of Most Bismuth Recovered
Not indicated.....	0.109	0.107	+0.002	+ 2		
Not indicated.....	0.105		−0.002	− 2		
Not indicated.....	0.101		−0.006	− 5		
Bottom of flask..	0.09		−0.017	−16		
Middle of flask...	0.133		+0.026	+25	Bottom	Middle
Top.....	0.123	0.127	−0.005	− 4		
One-half full.....	0.135		+0.008	+ 7		
Bottom.....	0.124		−0.003	− 2	Top	One-half full
Top.....	0.121	0.106	+0.015	+14		
Bottom.....	0.092		−0.014	−13	Bottom	Top
Top.....	0.127	0.119	+0.008	+ 6		
Below half.....	0.131		+0.012	+10		
Bottom.....	0.099		−0.02	− 1.7	Bottom	Almost empty
Top.....	0.086	0.107	−0.021	−19		
Bottom.....	0.129		+0.022	+20	Top	Bottom

digestion of the preparation with nitric acid, precipitation of the bismuth with hydrogen sulphide and gravimetric determination of the metallic bismuth. For the tartroquiniobine the method used was that employed by the American Medical Association chemical laboratory, the oil first being extracted by petroleum ether and the dry powder oxidized by cold nitric acid.

The actual amount of bismuth in the different samples varied widely. The variation in one flask was from —16 to +25 per cent, or a total of 41 per cent. In another there was a variation from —19 to +20 per cent. The median variation was ±10 per cent. The salt is in an oily suspension, and the difference in dosage is probably not greater than the difference in absorption, and therefore it may mean little therapeutically. One large dose might counterbalance the next one, which might be smaller. The fact remains, however, that an average injection does not give the expected dose.

There seemed to be no great difference in dose between samples from the top and bottom. In two bottles the greatest bismuth content per cubic centimeter was found in the middle, in one at the top, and in two others at the bottom. In taking these samples the blunt tip of the record syringe was plunged into the bottle after shaking, the dose was removed, the needle inserted and the dose administered as collected. The differences obtained have no relation to the quantities of suspension remaining in the bottle under the conditions of our observations. They

TABLE 2.—*Analysis of Bismuth Salicylate*

Level of Liquid in Bottle	Bismuth, Gm. per Cc.	Average of Samples From Bottle	Variation From Average	Variation, per Cent	Level of Least Bismuth Recovered	Level of Most Bismuth Recovered
Salicylate cream:						
Not indicated...	0.08					
	0.095					
Salicylate in oil:						
Top.....	0.051	0.056	—0.005	— 8		
Top.....	0.063		+0.007	+12		
Three-fourths full	0.055		—0.001	— 1	Three-fourths full	Top
Top.....	0.054	0.065	—0.011	— 2		
One-third full.....	0.073		+0.008	+12		
One-fifth full.....	0.069		+0.004	+ 6	Full	One-third full

TABLE 3.—*Analysis of Tartroquiniobine* *

Level of Liquid in Bottle	Total Weight of Compound, Gm.	Difference From Standard (0.208 Gm.)	Difference, per Cent
Top: little or no shaking.....	0.049	—0.158	—76
Top: vigorously shaken	0.0717	—0.135	—65
Below top: routine shaking.....	0.085	—0.122	—58
Middle: routine shaking.....	0.085	—0.122	—58
Bottom: routine shaking.....	0.275	+0.068	+32
Sediment	0.3935	+0.1865	+90
	0.3838		

* According to the American Medical Association chemical laboratory, a 2 cc. dose contains 0.06 Gm. of metallic bismuth and 0.2084 Gm. of dry substance.

must therefore be due to variation in the amount of shaking, which evidently is not uniform when performed by hand. The average amount of metallic bismuth found in the bottom of the bottle was 0.128 Gm., while that in the rest of the bottle was 0.121 Gm.

In the salicylate in oil the range of variation is not so great as in the tartrate, though in one bottle it was from +12 to —8 per cent. The smaller variation may be due to the fact that the preparation is of smoother suspension. The only two samples of salicylate cream obtained showed little or no variation.

In the analysis of the tartroquiniobine, the total dry substance in suspension was weighed, after washing with petroleum ether. As table 3 shows, the sample taken from the top layer contained but a fourth of the theoretical dose of dry substance if the bottle was shaken lightly; even when it was shaken vigorously, the top sample contained only a third and the sample from the middle, two fifths of the dose, while the bottom sample contained a dose and a third, and the sediment sample nearly a double dose. These differences are too large to be considered therapeutically negligible.

Since the amount of total dry matter varied so greatly, it appeared probable that the ratio of the two bismuth compounds might also vary. Accordingly, another bottle was shaken more vigorously than in routine clinical use, and the upper half decanted from the lower. The quantity of dry material in 2 cc. samples of the two halves was determined as already described; then one set of samples was extracted with acetone, water and concentrated and dilute hydrochloric acid. The decreases of weight in the successive operations correspond to bismuth iodoquinine, to sodium potassium bismuth tartrate and to impurity, presumably bismuth hydroxide. The ratio of the weight of bismuth iodoquinine to the sodium potassium bismuth tartrate should be 2.25 : 1. The ratio actually found in the upper layer was about 8 : 1; in the lower layer, about 1.7 : 1. It is evident, therefore, that the ratio of the two compounds in the layers varies even more widely than the total dry weight. The actual quantities (in grams) determined in this way were as follows:

	Bismuth Iodoquinine	Sodium Potassium Bismuth Tartrate	Bismuth Hydroxide
Top	0.112	0.0146	0.004
Bottom	0.214	0.126	0.004
Theoretically they would be	0.144	0.064	0

Corresponding to this, the bismuth content of the dry material from the top was 25 per cent, and of that from the bottom, 36 per cent, whereas the bismuth content of 2 cc. was theoretically 30 per cent.

Thus the examination of the dry material extracted from tartroquiniobine, even when the bottle was shaken vigorously, revealed that a dose from the top sample was but one third of a dose and that from the sediment nearly a double dose. Moreover, the ratio of the two compounds, bismuth iodoquinine and sodium potassium bismuth tartrate, varied even more widely than did the total dry weight—a double reason why this preparation in bulk is not to be relied on.

We believe that our limited investigation justifies the following conclusions:

1. There may be variations as great as 41 per cent in dosages taken from the same bottle of bismuth salt suspension, and with one prepara-

tion even 90 per cent; if one considers the sediment in the tartroquiniobine, it may arise to as great an amount as 166 per cent. In other words, one does not always give the dose one expects. The variation was least marked with a bismuth salicylate cream; it was greater with an oil suspension of bismuth subsalicylate and still greater with a potassium bismuth tartrate. The greatest variation was in a suspension of sodium potassium bismuth tartrate iodobismuthate of quinine in camphorated oil. Moreover, this shows an even greater variability in the ratio of the two bismuth compounds in samples from the top and from the sediment in a 25 cc. bottle.

2. Other things being equal, soluble solutions of bismuth salts will allow of more exact dosage. This refers to exactitude of dosage only, the question of type of salt not entering into this consideration.

3. If a more exact dosage is important, it could perhaps be secured by the use of a mechanical mixer.

ABSTRACT OF DISCUSSION

Dr. Howard Fox, New York: I was interested to hear that the smallest variation in dosage was found in a bismuth salicylate cream; in other words, in a preparation having a more solid base than an oil. I should like to ask Dr. Cole whether he has examined the preparation of bismuth in a cream put up in so-called "collapsules." Such a preparation is at least convenient. I have in mind the remarks of another essayist regarding the wisdom of making antisyphilitic treatment as simple as possible, provided it is efficient.

Dr. Albert Keidel, Baltimore: I wish to ask Dr. Cole if these preparations contain glass beads. In my experience with the glass beads one is not likely to get much sedimentation. With a fresh bottle it is necessary to carry out vigorous shaking for some minutes, for the preparation may have been standing on the drug store shelf for some time. However, in my experience with adding glass beads, which we always do if they do not come in the preparation, at the bottom of the bottle there is very little thickening of the oil suspension.

Dr. Joseph Grindon, St. Louis: Dr. Cole has drawn our attention to an important matter. I wonder if what he has learned in regard to bismuth does not apply to all nonsoluble solutions, the mercurials, for example, in the treatment for syphilis. Twenty years ago, Hallopeau and Fouquet (Traité de la syphilis, p. 94) made the statement that there were about fifty deaths on record from intramuscular injections of insoluble mercurials. What Dr. Cole has said is a reason for preferring the soluble preparations. Then there is the danger of embolism and the disagreeable nodules left after the use of insoluble preparations, and now comes the question of the uneven distribution of the drug.

Dr. Earl D. Osborne, Buffalo: In regard to using bichloridol and bismudol in collapsule tubes, we used them for a number of years, but finally gave them up. As you all know, the drugs in these collapsules are soluble salts of mercury and bismuth. About two years ago it was shown in the laboratories of the American Medical Association that the gelatin wall of the collapsule took up a large percentage of the mercury. After using bismudol for over two years we gave it up because, with 3 grain (0.19 Gm.) doses, we were unable to secure a single case of intolerance to bismuth; nor did we produce a single bismuth line on the gums.

Dr. Frank W. Cregor, Indianapolis: I think that Dr. Osborne is in error regarding the work of the American Medical Association on the absorption of bismuth by the wall of the tubes. What the American Medical Association did was to examine ampules of bichloridol and report that some of these ampules, at least, contained a negligible quantity of mercury.

Acting on this information, I asked Dr. Harger, of the Indiana University School of Medicine, to examine some of these ampules, and on doing so he reported that, in his opinion, the mercury had passed from the vehicle in the ampule to the gelatin wall with which it was lined; on examination of this lining wall membrane, he found mercury.

On the basis of this information, the bichloridol product was withdrawn from the market.

I do not think that this examination applied to bismuth. If it did, I am in error.

Dr. Harold N. Cole, Cleveland: Of course, the criticism that has already been mentioned would apply to the collapsible tubes. It would be impossible to use them in a clinic where one gives from seventy-five to a hundred injections in an afternoon. It would be applicable if one had time, but time is an element to be considered. It would be effective, but if I were using any preparation in a collapsible tube, I would want to investigate it very thoroughly.

In regard to the glass beads, the thing that Dr. Keidel spoke of would apply. We shook the preparations thoroughly, but a dose taken one third of the distance from the top of the bottle was about one third of what it should have been.

FATAL IODODERMA*

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This is a report of a fatal case of iododerma that presented many unusual features. The eruption consisted of macules, papules, tubercles, rupioid lesions and fungating and granulomatous ulcerations. Involution occurred at any stage in all types of lesions. Four months after the eruption began, which followed a few weeks of medication with potassium iodide, the patient died of profound intoxication. For four weeks preceding death, the patient passed large quantities of iodides in the urine. The chemical examination made post mortem also revealed a large amount of iodides in the skin, liver and kidneys.

Many dermatologists [1] examined this patient at one stage or another while he was under our care, and most of them concurred in the clinical diagnosis of an eruption due to iodide. A few, however, considered the following conditions: atypical eruption of mycosis fungoides, rupioid syphilis and pemphigus foliaceous. There was a great diversity of opinion among the pathologists who made microscopic studies of sections taken from the lesions as to whether the atypical proliferation of cells was a neoplastic or an inflammatory process.

While there are numerous reports in the literature of deaths resulting from the bullous type of eruption due to iodide, only a small number of deaths from nodular and tuberculous iododermas have been reported.

The following cases are referred to because of certain features described which simulate those found in the case reported here.

Besnier's case,[2] reported in 1882, consisted of "veritable tumors ranging in size from that of an ordinary pea to that of a chick-pea, copper red, flabby, almost fungous and with punctate lacunae analogous to those of an anthracoid furuncle."

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* Submitted for publication, Feb. 8, 1931.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

* From the Department of Dermatology and Syphilology of the New York Post-Graduate Medical School and Hospital.

1. Manhattan Dermatological Society Transactions, Arch. Dermat. & Syph. **23**:365 (Feb.) 1931; ibid. **23**:579 (March) 1931.

2. Besnier, E.: Anthracoid Acne Due to Iodides, Ann. de dermat. et syph., 1882, p. 171.

Hallopeau's case,[3] in 1888, presented vegetating pemphigus-like lesions, some of which were the size of a 50 centime piece with vegetations projecting for several millimeters.

In 1890, Jonathan Hutchinson[4] reported the case of a man, aged 26, who received potassium iodide over a period of ten weeks. During this time an eruption appeared. The dose was increased, and the eruption become more pronounced, with large vegetating and tuberous lesions. Iodides were discontinued, but the patient died fourteen days later. Hutchinson had seen two similar cases previously, and gave the opinion at that time that undoubtedly many such cases had been diagnosed as mycosis fungoides or as syphilis. Pollitzer saw the patient whose case is reported here, and immediately remarked on its resemblance to Hutchinson's case.

Later, Hutchinson[5] (in 1907) reported a case as "iodide sarcoma" in which there were solid, opaque lesions ranging in size from that of a pea to that of a nut, which developed sharply circumscribed ulcerations.

Walker[6] reported a case in 1892 with soft, dark yellowish and brownish tumors, some of which projected for 8 mm.

Fordyce[7] reported three unusual cases in 1895. In the first there were deep subcutaneous nodules the size of an egg, which enlarged on ingestion of potassium iodide and disappeared when the drug was discontinued. The second presented dime-sized, brownish-red tubercles, which became enlarged and developed dark red superimposed crusts. The last two cases he described as typical anthracoid forms of iodide eruption.

In 1896, Canuet and Barasch[8] reported an unusual case in which there were many pea-sized papules and pustules over the face and scalp and bullous lesions over the hands and wrists. On each cheek there was a large, irregular, dark red tumor, with thick edges, which measured 5 cm. in diameter. The summit of the tumor was truncated. There were several ulcerated, vegetating lesions on the chin, and on

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3. Hallopeau, H.: Vegetating and Atrophic Form of Pemphigus Referable to Iodine, Ann. de dermat. et syph. **9**:286, 1888.

4. Hutchinson, J.: A Case of Iodide of Potassium Eruption, Arch. Surg. London (Appendix), 1890, vol. 1.

5. Hutchinson, J.: Iodide Sarcomata, Brit. J. Dermat. **19**:441, 1907.

6. Walker, Norman: Vegetating and Condolomatous Iodide Eruption, Lancet, May 12, 1892.

7. Fordyce, J. A.: Notes on Drug Eruptions, J. Cutan. Dis. **13**:496 (Dec.) 1895.

8. Canuet and Barasch: A Fatal Case of Malignant Iodism of the Mycotic Type, Arch. gén. de méd. **2**:424, 1896.

the neck there was an ulceration 2 cm. in size. The condition was first diagnosed as mycosis fungoides, but Fournier made the diagnosis of an eruption due to iodides or bromides, and a few days later iodides were found in the urine.

Neumann [9] reported a case in 1899 in which there were tuberous lesions of the face and extremities following medication with potassium iodide for six days. Iodides were present in the urine. Death occurred five weeks later from profound intoxication. At death, there were many large, ulcerating tuberous lesions and smaller lesions with a proliferating wall and central ulcerations. Post mortem, similar lesions were found on the gastric mucosa.

In 1899, Hyde [10] described three cases of the tuberculous type.

Rosenthal,[11] in 1901, reported an unusual case which he described as "iododerma tuberosum fungoides" occurring in a patient aged 29. Potassium iodide had been administered for asthma at intervals over a period of years. Papulopustules had developed once previously, and had healed promptly when the drug was discontinued. The tumors were soft and covered with crusts and were limited largely to the face. The histologic picture of one of the facial tumors presented the following changes: There was a diffuse infiltration of the corium consisting of closely packed polymorphonuclear and mononuclear leukocytes surrounded by markedly edematous tissue. There was a marked proliferation of the epidermis; the basal and prickle layers showed abundant mitoses. There were hypertrophy of the granular layer and an irregular detachment of the corneous layer. There were several abscesses in the corium, some of which had broken through the epidermis. The sebaceous and sweat glands were not directly involved.

Rosenthal expressed the belief that iododerma represents an inflammatory granulation tumor associated with hyperkeratosis and parakeratosis.

Danel [12] reported a fatal case of bullous and vegetating iododerma in 1926, which presented papular, bullous and vegetating lesions on the face, neck and extremities, some of which resembled pemphigus vegetans.

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9. Neumann, J.: A Peculiar Type of Eruption of the Skin and of the Gastric Mucosa Due to Iodine, Arch. f. Dermat. u. Syph. **48**:323, 1899.

10. Hyde, J. N.: Tuberculous Form of Iodide Eruption, Arch. Dermat., 1899, p. 333.

11. Rosenthal, O.: Iododerma Tuberosum Fungoides, Arch. f. Dermat. u. Syph. **57**:3, 1901.

12. Danel, L.: A Fatal Case of Bullous and Vegetating Iodine Eruption, Bull. Soc. franç. de dermat. et syph. **33**:40, 1926.

REPORT OF CASE

History.—H. H., aged 31, white, married, was first seen on Sept. 3, 1930. His health had been good with the exception of pain over the region of the right kidney eighteen months previously. The urologist who took care of him at the time diagnosed this as renal colic. There have been no symptoms since. There had not been any lesions of the skin until one year before, when brownish, slightly scaly patches, which gave no subjective symptoms, appeared on his legs. In June, 1930, he consulted a physician for the treatment of these lesions, and a tonic was prescribed which contained potassium iodide and arsenic. The patient took three

Fig. 1.—Face of the patient seen on Sept. 15, 1930, showing soft, fleshy papules, some of which had become crusted.

doses daily of 5 grains (0.325 Gm.) of potassium iodide and 1/60 grain (1 mg.) of arsenic for four months. Shortly after, many small, bright red papules and nodules developed over the trunk and extremities. The patient had also been taking iodized salt on his food for several years. (There is 1 mg. of iodide to each gram of iodized salt.)

Physical Examination.—The patient was a well nourished, well developed white man, weighing 143 pounds (64.9 Kg.). Except for the lesions on the skin, the general physical examination gave essentially negative results. The temperature was 100.1 F. The patient looked worried. His face and neck presented a moderate

number of bright red, soft, fleshy papules and nodules varying in size from 6 to 12 mm. in diameter and rather characteristic of iododerma (fig. 1). There were similar but larger lesions on the scalp with dry, yellowish crusts on them and others which were moist. On the trunk and extremities, there were lesions in various stages of evolution and involution. There were a moderate number of bright red macules ranging in size from that of a pinhead to that of a pea; a number of the latter were infiltrated and slightly raised. Some of the larger and more infiltrated lesions became scaly over their central portions. Numerous erythematous papules and nodules, varying in size from 12 to 25 mm. in diameter, were scattered all over

Fig. 2.—Trunk and extremities on Sept. 15, 1930. Note the lesions in various stages of development.

the foregoing areas, especially the chest and back (fig. 2). The small papules had a grayish scale over the central portion, and most of the larger papules were covered with thick, laminated, oyster shell-like, brownish, rupioid crusts. There were several yellowish-brown, slightly scaly patches over the lower extremities, which varied in size from 15 to 40 mm. Over the middle third of the left tibia, there was an ulcerated granulomatous lesion about 3 inches in diameter and a similar larger one on the left buttock. There were no lesions in the mouth or on the palms and soles. There was no adenopathy.

Progress of the Case.—With the discontinuance of the iodides, no new lesions appeared for about ten days, and all lesions present were undergoing involution.

Then, from time to time, there was an eruption of new, dark red macules, and on a few occasions there was a shower of hundreds. Some of these appeared and disappeared in twenty-four hours; others increased in size and became elevated. In this stage some of the lesions underwent involution and disappeared, while others continued to increase in size and became tuberous. While the majority of these nodules ranged in size from 10 to 25 mm. and were covered with grayish scales, some were from 40 to 60 mm. in diameter and were covered with thick, laminated, oyster shell-like crusts which were the result of the gradual heaping up of scales as the lesions became larger (fig. 3). The process of involution occurred in some of

Fig. 3.—Condition on November 7. Note further development of the lesions into large crusted nodules on the face and ulcerated lesions on the chest.

the lesions irrespective of the stage of development. When the smaller lesions disappeared, the skin of their former site resumed a normal appearance. When the large crusted lesions underwent involution, the crusts became more dry and dropped off, leaving a smooth pigmented area, and in a few days the skin assumed a normal appearance. By the third week the large, granulomatous, ulcerating lesion on the left buttock had disappeared, leaving an apparently normal skin. The similar lesion over the left tibial region increased in size and was present at death. While some of the larger crusted nodules underwent involution with a drying of the laminated crusts which dropped off leaving an apparently normal

skin, in other lesions the crusts fell off leaving large vegetating excavated ulcers. The progress of evolution and involution of the large lesions took from six to eight weeks or more. Numerous lesions continued to increase in size and become ulcerated, the exudation and dry crusts of which had an obnoxious odor. One large nodule appeared on the left lower lid and closed the eye. Two weeks before death, almost the entire surface of the skin was covered with the large, vegetating, ulcerating and rupioid lesions (fig. 4). As more of the surface of the body became covered with lesions, the patient's temperature rose to 104 F. and became irregular. A few days before death, the patient became delirious and comatose. He died on Nov. 17, 1930. (A general physical examination gave negative results until a few days before death. At no time had there been lesions in the mouth.) There never was any adenopathy. Various therapeutic measures were instituted, such as intravenous injections of saline, sodium thiosulphate, dextrose, transfusion of blood, etc., without apparently influencing the development of new lesions.

Laboratory Examination.—The blood counts, made on four different days, are shown in the table.

Patient's Blood Counts

	Blood Counts								
	Red Blood Cells	White Blood Cells	Small Lymphocytes	Large Lymphocytes	Polymorphonuclear	Eosinophils	Basophils	Mononuclear	Hemoglobin
October 20	4,500,000	23,750	16%	4%	76%	....	....	4%	85%
October 27	.....	19,950	10%	4%	80%	....	....	....	....
November 2	.....	18,100	7%	....	83%	....	....	8%	....
November 6	4,550,000	17,500	9%	3%	81%	2%	....	5%	100%

On November 5, chemical examination of the blood showed: urea nitrogen, 13.4; sugar, 145; carbon dioxide combining-power, 52.2.

A culture of the blood on October 25 was negative after forty-eight and ninety-six hours.

On September 13, October 2 and October 10 the Wassermann test with the Kolmer and cholesterolized antigens, and the Kahn and Kline tests were all negative.

On October 20, dark-field examinations of the serum from several lesions were negative for Spirochaeta pallida. Fontana stains for Spirochaeta pallida from serum were negative. Serum and sweat with starch-litmus paper gave negative tests for iodine.

On November 3, cultures from the lesions of the skin contained Staphylococcus aureus and albus.

On November 14, cultures from the lesions of the skin contained predominant Staphylococcus aureus and few Staphylococcus albus.

On November 17, phytopharmacologic examination of the blood serum by Dr. Pels of Baltimore gave a reading of 65 per cent (suggestively toxic).

Routine urinalyses were essentially negative throughout the patient's illness, except for slight traces of albumin shortly before death. Iodides were present on many occasions in a later stage of the illness.

Two attacks of hematuria occurred within twenty-four hours unaccompanied by pain or other urinary symptoms. The urologic examination was entirely negative; the urologist's impression was that the hematuria resulted from the passage of a stone, as the patient had had a previous attack of renal colic.

Special examination of the urine for iodides revealed the following results:

September 6	Negative for iodides
September 15	Negative for iodides
October 1	Negative for iodides
October 21	Positive for iodides (qualitative method)
October 27	Positive for iodides (qualitative method)
October 29	330 cc. of urine showed 29.7 mg. of iodides
October 31	970 cc. of urine, 0.92 mg. of iodides
November 5	Iodides present
November 14	0.28 mg. of iodides per 1,000 cc.
November 15	0.044 mg. of iodides per 1,000 cc.
November 16	0.031 mg. of iodides per 1,000 cc.

Normally there are 25 mg. of iodine in the entire body, 80 per cent of which are in the thyroid gland.

Fig. 4.—Condition on November 7, ten days before death, showing lesions in all stages on the trunk and extremities. Note the numerous large oyster-shell-like lesions and others which had become ulcerated.

Postmortem Examination.—The results of the postmortem examination were negative except for terminal changes preceding death, i. e., bronchopneumonia, congestion of the liver, spleen and kidneys. The chemical examination of the tissue post mortem showed iodides in the following amounts: skin, 0.2 mg. per hundred grams; liver, 0.022 mg.; kidneys, 0.018 mg.

Drs. W. J. MacNeal, J. Ewing, D. Satenstein, A. Frazer, J. F. Frasier, Paul Klemperer and S. Pollitzer made the histologic examinations of the skin lesions; on the studies of Drs. P. Klemperer and S. Pollitzer the following histologic report is based:

The earliest lesions examined were approximately 5 mm. in diameter, slightly elevated and infiltrated. The lesions in this stage showed a horny layer, largely desquamated, and an epidermis that was disorganized by intracellular edema; in some places the cells were widely separated, forming cavities which were partly filled with cellular elements: irregularly round or polygonal mononuclear cells

about twice the size of a red blood cell, with apparently ameboid cytoplasmic processes; occasionally faintly basophilic; large pale nucleus with distinct nuclear membrane and one or more nucleoli. In the papillary and subpapillary layers there were the changes usually seen in any severe toxic process, i. e., edema and swelling of the endothelial nuclei, etc. Throughout the cutis there were aggregations of cells about the capillaries which will be described later.

Further advanced lesions showed more marked changes in the epidermis, and little of the epidermis remained. Throughout the cutis there was a dense infil-

Fig. 5.—Biopsy section from a well developed papule showing diffuse cellular infiltration throughout the cutis and vesiculation and destruction of the epidermis. Hematoxylin eosin stain; reduced from a magnification of × 120.

tration which consisted mostly of cells of an unusual character, so much so that it was considered to be a neoplasm by several of the pathologists, most of whom at the time of their microscopic studies had not been informed of the clinical course of the disease. It was only after careful study of the histology of the earliest lesions that the cells found in the dense infiltration were recognized

as belonging to the group of mesenchymal cells described by Marchand as adventitial; cells generally considered as histiocytes or reticulo-endothelial cells of Aschoff. The adventitia of the capillaries showed a great increase in the number of these cells, many presenting mitoses, and a large number of these cells were recognized throughout the infiltration.

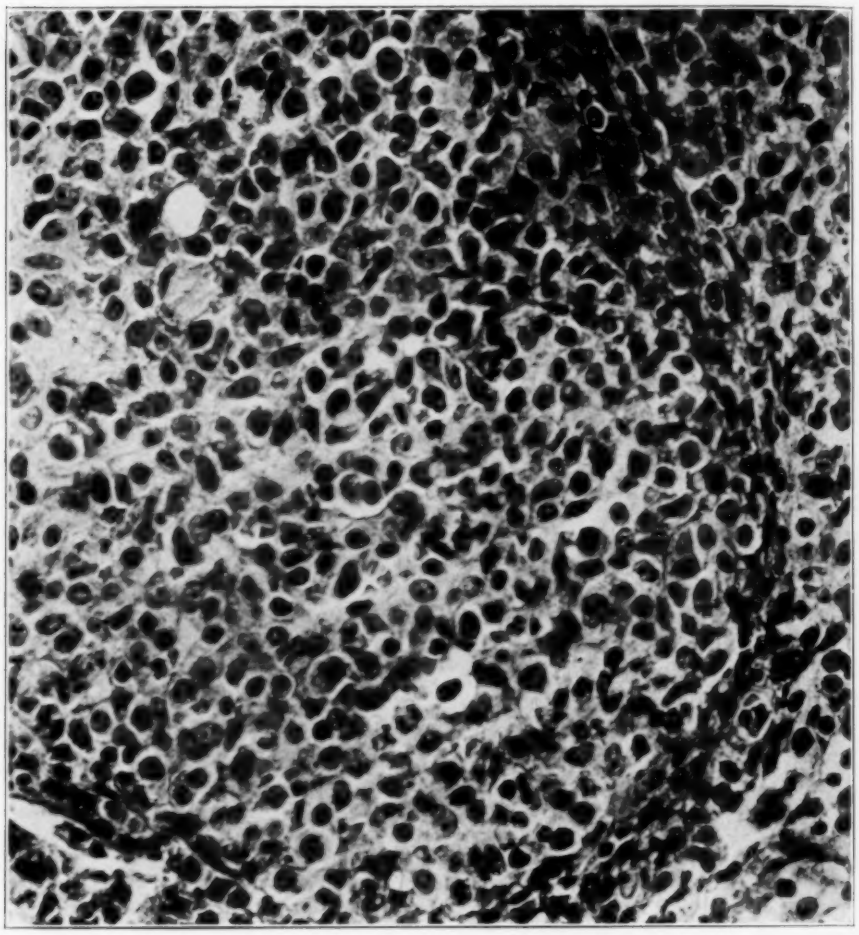

Fig. 6.—High power magnification of figure 5 showing diffuse infiltration of large cells (adventitial cells) and many mitotic figures. Hematoxylin eosin; reduced from a magnification of × 280.

The adventitial cells of Marchand are mesenchymal cells showing a large nucleus with two or more nucleoli and a narrow zone of cytoplasm that stains very feebly with basophilic dyes and often may be seen to run out into a caudate process. These cells are recognized as having the characteristics of embryonal cells and they may develop along several lines of evolution, i. e., connective tissue cells, etc.

The great mass of infiltration which fills the entire cutis was made up of these cells which in places even penetrated into the epidermis.

There were few polymorphonuclear cells in the upper portion of the later lesions. In addition, the later lesions contained numerous fibroblasts with greatly swollen nuclei.

The entire picture was that of a blood-borne irritative process with the unusual feature of an enormous proliferation of the histiocytic cells which emanated from the adventitia of the capillaries.

After the postmortem examination, practically all of the organs were examined microscopically and, with the exception of the terminal bronchopneumonia of both

Fig. 7.—Nodule from the skin at autopsy showing epidermic and subepidermic vesicles with diffuse infiltration in the entire cutis. Van Giesen stain; reduced from a magnification of × 120.

lower lobes and hyperplastic changes in the thyroid gland, were found to be normal. Dr. Klemperer was of the opinion that the hyperplasia of the thyroid gland was due to the long continued ingestion of iodine and was the type described by Kocher as "iodide thyroid."

The lymph nodes were not examined, as no macroscopic changes were present at the time of the autopsy.

Deductions.—It seems reasonable to assume that the toxic agent in this case was the iodine; it had been ingested in minute doses over several years, and there was a sudden eruption of lesions following the ingestion of larger doses.

The remarkable feature in this case, from the pathologist's point of view, was that the chemical or toxic effect was manifested by a tremendous proliferation of cells, which appeared to be embryonal mesenchymal; these cells were so numerous that one of the pathologists termed the lesions reticulosis tumors.

Fig. 8.—High power magnification of figure 7 showing infiltration of adventitial cells. Van Gieson stain; reduced from a magnification of × 280.

SUMMARY

1. A fatal case of eruption due to the ingestion of iodide with unusual clinical features has been reported.

2. Microscopic studies revealed a peculiar inflammatory process which was difficult to interpret, and which in some stages was suggestive of a neoplasm.

3. A study of the rapid evolution and involution of the lesions as described, with the finding of large amounts of iodides in the urine, skin, kidneys and liver, could only lead one to the conclusion that the eruption was due to the iodides.

4. It can easily be conceived that, following the ingestion of iodized salt for some years, the patient in the case reported was sensitized to such an extent as to develop a fatal iododerma after the ingestion of a tonic containing small amounts of potassium iodide. As other cases are on record of eruptions from the use of iodized salt, even though not severe, we believe that the indiscriminate use of iodized salt should be avoided, and that the medical profession should warn the public of this danger.

100 West Fifty-Ninth Street.

MULTIPLE LESIONS OF THE SKIN APPARENTLY OF EPITHELIAL ORIGIN RESEMBLING CLINICALLY MYCOSIS FUNGOIDES

REPORT OF A CASE *

LLOYD W. KETRON, M.D.
AND
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BALTIMORE

History.—A F., aged 59, white, a bricklayer, was admitted to the clinic of the Johns Hopkins Hospital on Feb. 24, 1930. He complained of a disorder of the skin, the first signs of which he had noticed in the spring of 1928. There had never been a similar complaint in any member of his family. His paternal uncle, a sister and his mother died of tuberculosis, and he had been exposed to infection from his mother for a number of years. So far as his own past history was concerned, he had had measles and whooping cough in childhood and typhoid fever at the age of 25. At the age of 20 he had a urethral discharge, which cleared up in two weeks by the use of injections. For several years he had been deaf in the left ear, and the last of his teeth were extracted some time ago. He had been married for twenty-five years; his wife had had no miscarriages and was well at 57 years of age. He had two children, both of whom were living and well. Prior to the onset of his present illness, his general health had been good, and he had never had any disease of the skin.

The onset of the present illness, about two years before he came to the clinic, was preceded by no change in the mode of life nor by any unusual occurrence. At that time he noticed on the flexor surface of the left wrist a round, copper-red area, approximately 2 cm. in diameter, which was not tender or painful and did not itch. This area persisted for about eight months, during which time it grew slightly larger and "thickened up a bit." It then began to fade from the periphery toward the center. About six months after its appearance, a bright red ring developed on the extensor surface of the upper part of the left arm, just above the elbow. It was somewhat thickened, and surrounded a central clear space about 1.5 cm. in diameter. The red color gradually spread inward and outward until the original circular outline was lost in a solid red erythematous area which was somewhat larger than the area at first included. Before it had fully developed, others began to make their appearance on the arms and legs, and since then there had been a gradual progression of the disease with the development of nodules, some of which had ulcerated. Some of the lesions had disappeared, but the patient thought that most of them had persisted. Physically, he had felt quite well, and there had never been any itching.

Shortly after the onset of the disease, the patient had consulted a physician who had treated him for "ringworm," with liquid applications. Later he saw another physician, who diagnosed the condition as psoriasis and gave him five intravenous injections of what he thought were arsenicals. Salves were also prescribed, but he was benefited only slightly, if at all, by the treatment. From that

* Submitted for publication, Feb. 8, 1931.

* From the Johns Hopkins Medical School, Department of Dermatology.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

time until the present examination he had used a great many proprietary ointments without obtaining any relief.

General Examination.—The patient was a healthy looking man, except for the condition of the skin. The mouth and pharynx were free from lesions. The teeth were absent, but the gums were in good condition. The tonsils were somewhat enlarged but not inflamed. Examination of the cardiovascular system revealed no

Fig. 1.—Side view of the patient showing below the iliac crest the circinate psoriasiform lesion from which the specimen for biopsy 3 was taken.

disease of the heart, but there were numerous extrasystoles and slight hypertension, the systolic blood pressure being 170 and the diastolic 95. There was a moderate pulmonary emphysema, but on roentgen examination the lungs were found to be normal. The liver was not enlarged, but the spleen was palpable. There was no enlargement of the lymph glands. Examination of the blood showed: red blood cells, 4,750,000; hemoglobin, 94 per cent; color index, 0.99;

white blood cells, 7,100, with a differential count of polymorphonuclear neutrophils, 64 per cent; eosinophils, 2 per cent; basophils, 1 per cent; lymphocytes, 24 per cent; mononuclears, 9 per cent. The platelets appeared normal. Analysis of the blood disclosed the following: nonprotein nitrogen, 36 mg. per hundred cubic centimeters; sugar, 100 mg.; uric acid, 3.1 mg.; total protein, 6.43 Gm. with an albumin-globulin ratio of 65:35. The Wassermann reaction of the blood was

Fig. 2.—Right hip and right thigh showing plaques, nodules and ulcers. The scars to the right of the large ulcer near the center of the photograph represent the location of a group of ulcers which the patient had when he first came to the hospital and which healed following the application of x-rays.

negative. Urinalysis showed a normal color, acid reaction, a specific gravity of 1.015, no albumin, no sugar and no blood, and was negative microscopically. Analysis of the contents of the stomach during fasting showed no free acidity, and 14 per cent combined acidity; after the ingestion of alcohol, the free acidity

and the combined acidity were both 8 per cent; after the administration of histamine, the free acidity was 48 per cent and the combined acidity 6 per cent. At the site of an intradermal injection of 0.1 cc. of 1:1,000 old tuberculin, there developed a reddened, slightly indurated area 1.5 cm. in diameter, a definitely positive reaction.

Fig. 3.—Urticarial and infiltrated plaques over the buttocks. This photograph was taken during the patient's last visit to the hospital, and there were many more lesions in this location than when he came for the first time.

The eruption, during the patient's three visits to Baltimore, consisted of several varieties of lesions located principally on the extensor surface of the arms and legs, on the lumbar region and the buttocks (figs. 1, 2, 3 and 4). The scalp, face, neck, upper portion of the back, palms and soles were not involved and never became affected while the patient was under our observation.

There were, in general, six types of lesions more or less promiscuously scattered over the involved areas: (1) Areas of simple erythema, smooth or slightly scaly, varying in size from 1 cm. in diameter to a large hand-sized area over the upper lumbar region from which the specimen for the first biopsy was taken. (2) Slightly raised, pinkish, roundish, annular or crescent-shaped, urticaria-like patches, which were most profuse over the lumbar region. These areas, however, were flatter and more plaquelike, and did not show the pinkish areola often seen in urticaria. (3) Roundish or crescent-shaped psoriasiform lesions. (4) Flat, roundish, or irregularly-shaped indurated plaques. (5) Nodules that varied

Fig. 4.—Large tumor on the left arm, which appeared after the patient's first visit to the hospital. The scar just above it represents the site of biopsy 4.

in size from a few millimeters to one on the left forearm that measured 3.5 cm. in diameter and 1 cm. in depth. The nodules were quite firm, of a reddish or normal skin color and had a smooth, scaly or slightly warty surface. Some of them had ulcerated. (6) Brownish-red or pigmented macules or patches which were thought to have followed lesions that had disappeared. In some of these areas, the skin showed a cigaret paper-like wrinkling which suggested that some atrophy had taken place.

It was not the rule for the various types of lesions to be sharply separated from each other. Over the lumbar region, particularly, the urticaria-like lesions showed a tendency to become confluent with various configurations. The psoriasiform patches in some cases occurred in or around the pigmented areas, and the nodules appeared in lesions of other character.

Treatment and Course.—During his first visit to Baltimore, the patient was under observation in the hospital from Feb. 24, 1930, to March 29, 1930. No special treatment was carried out, except three injections of antimony and potassium tartrate and suberythema applications of x-rays. At the time of his departure, there had been an almost complete healing of a group of ulcers on the right thigh, and some of the patches and nodules treated with x-rays had improved. On the other hand, there seemed to be more lesions present than when he was first admitted. He was given a solution of potassium arsenite to take while at home, and he again came to the clinic on May 16, about six weeks after his departure. At this time it was noted that many new lesions of all types had developed. There were several ulcerated nodules on the right thigh surrounding a scar which had resulted from a group of ulcers present in this location at his first visit. One

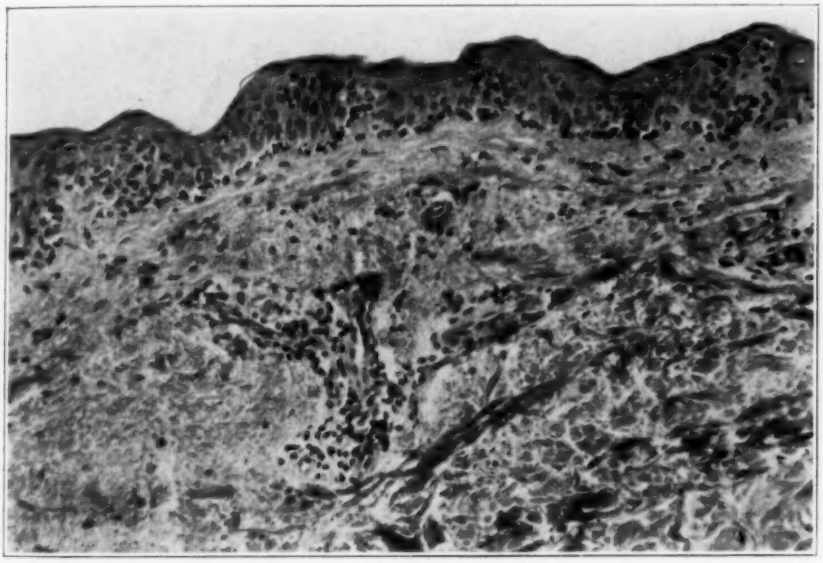

Fig. 5.—The edge of a section of the specimen removed for biopsy 4 showing the early stage of involvement of the epidermis. Note also the infiltration around the blood vessel in the corium.

skin unit of x-rays was administered to these lesions, and various other lesions were given from one-half to one skin unit. A blood calcium determination showed 11.1 mg. per hundred cubic centimeters. The blood count was: red blood cells, 4,888,000; hemoglobin, 93 per cent; white blood cells, 9,150, with a differential count of polymorphonuclear neutrophils, 59 per cent; eosinophils, 3 per cent; basophils, 1 per cent; lymphocytes, 34 per cent; mononuclears, 3 per cent. The bleeding time was two minutes, and the coagulation time was six and one-half minutes.

The patient's next visit was on July 10, 1930; his general condition was unchanged. Many new lesions, however, had developed, especially, the urticarial and plaque types, over the buttocks and lumbar region. Some of the ulcers that were treated with x-rays at the patient's last visit had healed, but other nodules

and ulcers had developed. The scalp, face, neck, palms and soles remained unaffected.

Histologic Study.—During the time that the patient was under observation seven biopsies were made from the various types of lesions. We have tried to trace the stages of development of the pathologic process from the earliest erythema to tumor formation rather than to describe each section in detail. The material was fixed in Zenker's fluid and solution of formaldehyde and stained with hematoxylin and eosin, Verhoeff's elastic tissue stain, polychrome methylene blue and special stains. The biopsy specimens were taken from these lesions: (1) A palm-sized area in the middle of the back showing only a faint, poorly defined

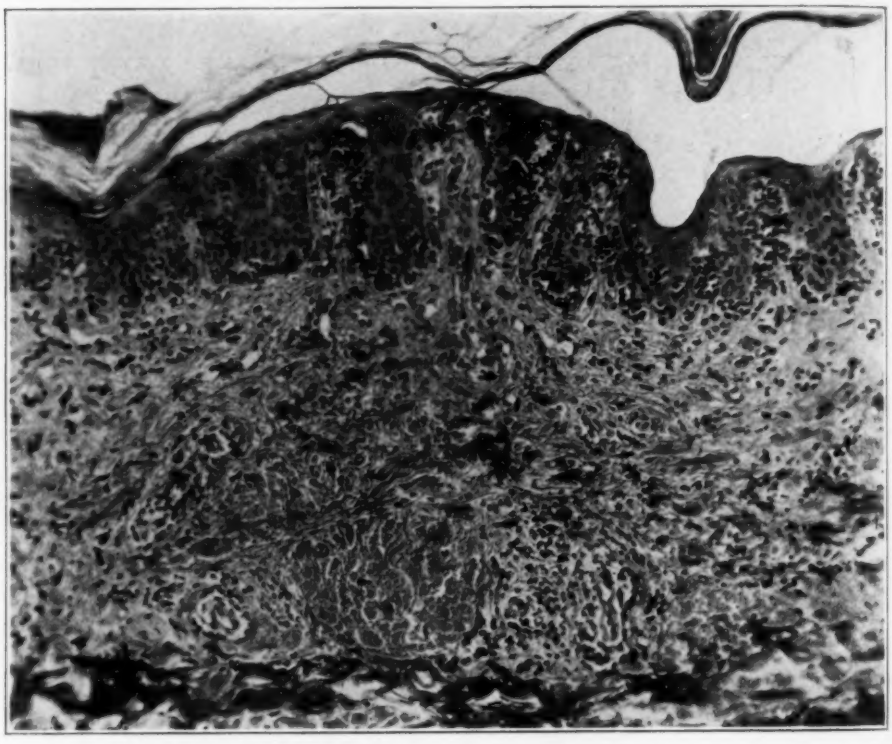

Fig. 6.—Specimen removed for biopsy 2 showing a stage of involvement of the epidermis more advanced than that in figure 5.

erythema. No other change was demonstrable clinically in the skin. (2) A faintly pinkish, moderately well defined, slightly elevated patch, 2 cm. in size, over the lumbar region. The surface was smooth and urticarial in appearance. (3) A nodule, 0.5 cm. in size, from the arm. (4) A moderately discolored, slightly scaly patch on the left hip, with a crescent-shaped, red, scaly, infiltrated thickened plaque about 1 cm. wide around one edge. The section was taken through this edge so as to include some of the skin on either side of the plaque. (5) A flat nodule, 1 cm. in diameter, on the calf of the right leg. The nodule was surrounded by a pigmented, slightly infiltrated, smooth zone about 1 cm. wide.

Only this latter tissue was used for histologic study, the nodule being utilized for other purposes. (6) A pea-sized nodule on the arm. The top of the nodule was cut off without a local anesthetic for supravital staining. (7) A scale-covered nodule, 1 cm. in size, on the arm. Macroscopically, after bisection, this lesion showed a thin, soft, jelly-like layer, just under the surface scale. Beneath this there was a whitish infiltrate, very tough and quite firm.

A. Stage of Simple Erythema (biopsy 1, serial sections): In the epidermis, we were unable to find any definite abnormalities. In the basal layer single cells were frequently seen showing shriveled nuclei in a clear ring of protoplasm, and occasionally larger vacuoles containing one or more pyknotic nuclei.

In the cutis, there was a mild perivascular infiltration in the papillary and subpapillary layers with dilatation of the vessels. This inflammation consisted prin-

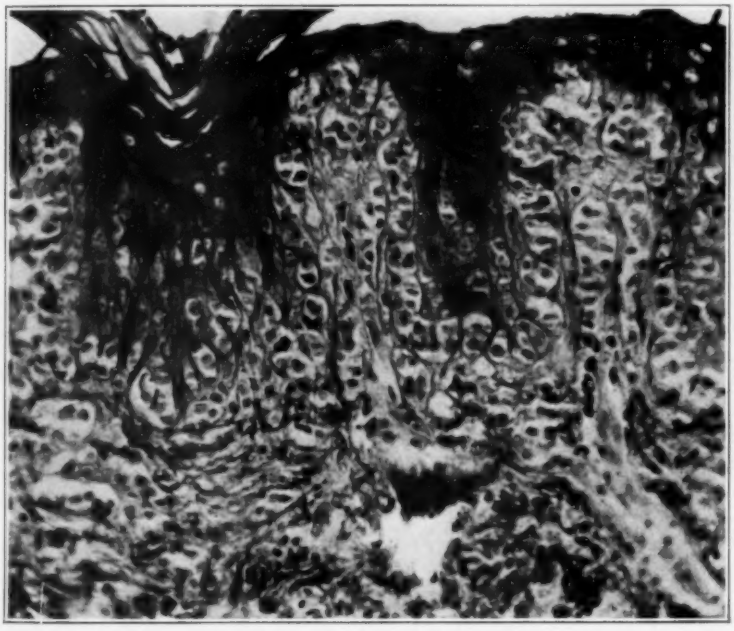

Fig. 7.—Section from the same biopsy as in figure 6, stained by MacCallum's bacterial stain showing how the basal cells occur in nests.

cipally of lymphocytes, endothelial or connective tissue cells and an occasional eosinophil. Mast cells were normal in number, and no definite change was found in the elastic tissue. A considerable chronic inflammatory type of infiltration was noted around one hair follicle, but this was thought to be most likely due to a seborrheic folliculitis. Otherwise, the adnexa of the skin were not affected.

B. Early Infiltrative Stage (biopsy 2 and tissue surrounding the small nodule of biopsy 4): Here the earliest changes in the epidermis appeared to be in the basal layer. Most of the nuclei were pyknotic and stained more or less diffusely black. The cytoplasm, to a great extent, had disappeared, leaving many of the nuclei singly or clumped in clear spaces (fig. 5). This row of cells, at first consisting of only one layer, appeared gradually to involve more of the epithelium until two or more layers or clumps of several cells were seen (fig. 6). The

tendency of the basal layer to proliferate in small cell nests was particularly well demonstrated in a section stained by MacCallum's bacterial stain (fig. 7). Here the bridges between the groups of cells were stained purple by the gentian violet of the stain, and one could follow the attachment of these bridges to the rete cells above and to the finer fibrils of the collagenous tissue below. As the process extended, larger masses of cells made their appearance, which displaced to a greater or less extent the epithelial pegs, or crowded into the papillae (fig. 8). While many cells showed shriveled black nuclei, in others the nuclei were larger and paler, and occasionally mitotic figures and nuclear fragmentation were present (fig. 8). In some of the pegs there had been a dissolution of intercellular sub-

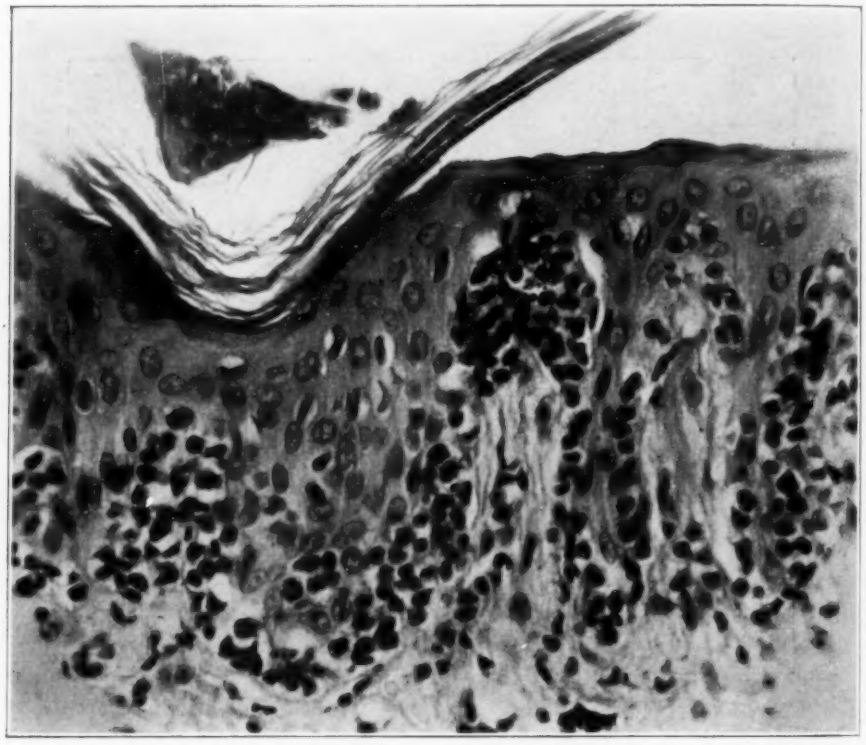

Fig. 8.—High magnification of the same section as in figure 6 showing the character of the cells. Note the group at the top of the papilla to the right of the center which has apparently pushed down the papillary body.

stance affecting only a small group of cells or almost the entire peg. The nuclei in some of these cells were pyknotic, others were larger and pale, and occasionally showed mitosis.

In the cutis, there was a considerable increase in cells apparently due to some small round cell infiltration, proliferation of fixed tissues and the wandering down of some of the tumor cells, principally from the papillae into which they had grown. These tumor cells lay in small nests, seemingly in lymph channels or in short rows between the connective tissue fibers. A few fragmented cells were observed. The hair follicles, sebaceous glands and sweat glands were also involved (figs. 9 and

10). Some of the sebaceous gland acini were almost completely replaced by tumor cells, some of which showed nuclear fragmentation and mitotic division. In the sweat glands changes in the cells occurred both in the secreting portion of the gland and in the excretory ducts. There seemed to be no degeneration of the elastic tissue.

C. Later Infiltrative Stage (psoriasiform plaque, biopsy 3): In this section, which was cut through a moon-shaped psoriasiform patch, including normal looking tissue on one end and slightly scaly pigmented tissue on the other, we were not able to decide which end belonged to the corresponding portions of the skin. Both ends showed changes somewhat similar to those already described, and the elastic tissue appeared normal on both sides. In the portion of the section corresponding

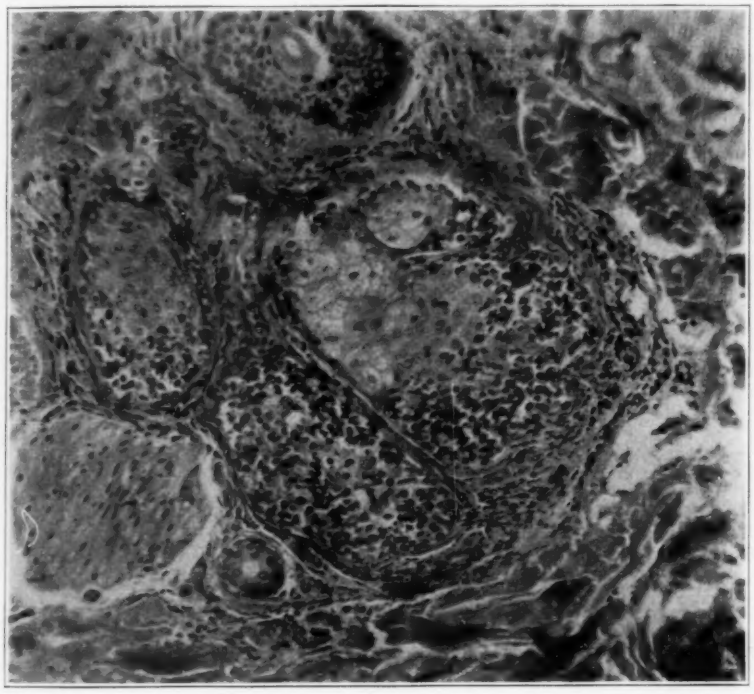

Fig. 9.—Hair follicle from the specimen removed for biopsy 2. Here may be seen a replacement of the sebaceous acini as well as a portion of the follicle by tumor cells. This process had apparently originated inside the capsules of these structures.

to the lesion, the outline of the epidermis was quite enlarged, and although its architecture was very much disarranged by changes in the epithelium, the lower border of the pegs was, in general, quite distinct (figs. 11 and 12). There were some hyperkeratosis and thickening of the granular layer. There was no well defined pyknotic basal layer as was seen in the tissue in biopsy 2, but a large portion of the rete was taken up by masses of tumor cells, separated by strands of the epithelial tissue. In some of the pegs where the changes were not so marked, the epithelium was swollen, and some of the nuclei were pyknotic and surrounded by clear spaces. In other areas there were a disarrangement of the cells and a

partial disappearance of intercellular substances. Most of the papillae were packed full of cells which in general showed smaller, more irregular and more darkly staining nuclei than those that filled the pegs (fig. 13). Mitotic figures and cell fragmentation were frequently encountered.

There was a considerable cellular infiltration in the cutis apparently composed of tumor cells as well as inflammatory cells. In this section no changes were found in the hair follicles, sebaceous glands or sweat glands.

D. Tumor Stage (biopsies 4, 5 and 6): In these three lesions there was solid tumor formation extending down to varying depths in the cutis, and partly, or in some areas completely, replacing the epidermis. The tumors were not sharply defined or encapsulated, but rows (fig. 14) or small masses of cells extended far out between the bundles of collagenous tissue or invaded the structures beneath.

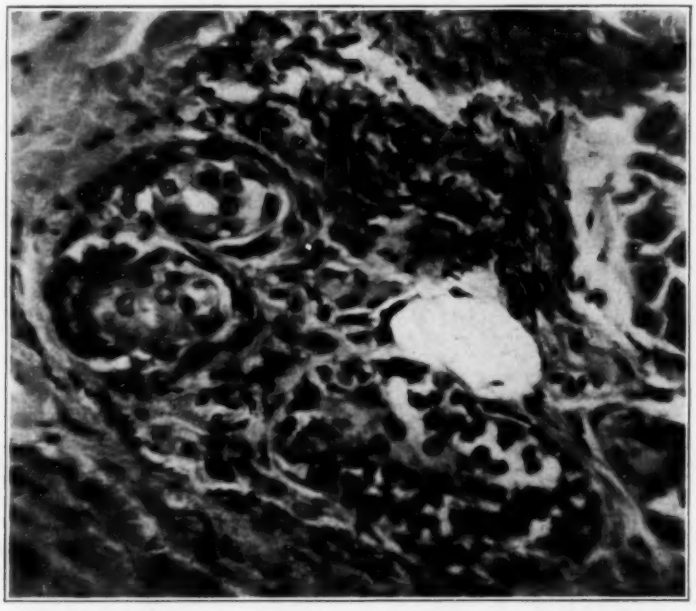

Fig. 10.—Sweat gland showing pyknosis of nuclei and dissolution of intercellular substances.

In some sections, the adnexa of the skin, which were quite removed from the border of the growth, were affected. The tumor cells were rather large, with roundish, oval or irregular nuclei and a thin, usually frayed, basophilic ring of cytoplasm. There was a moderately heavy network of chromatin, and the younger cells usually showed one or two large nucleoli. Mitotic figures were abundant. Toward the upper and older portions of the tumors, cellular fragmentation was extreme. Chromatin dust was scattered thickly everywhere, some of the particles being surrounded by a thin rim of cytoplasm. In some instances they were collected in small masses as if engulfed by phagocytes. A few fragmented cells were found here and there around the edges of the tumors where active invasion was in progress. A silver stain by Foot's modification of Bielschowsky's method, on a section of biopsy 7, showed a moderately dense reticulum of fine wavy fibrils running for the most part vertically from the surface of the tumor down until

they blended in with the coarser connective tissue bundles which had not been displaced by the tumor cells. Few blood vessels were present in the main part of the tumor.

E. Stage of Resolution (biopsy 7): The epidermis appeared as a narrow band in which most of the rete pegs and papillae had been replaced by tumor cells. There were some hyperkeratosis and thickening of the granular layer. The lower border of this epidermal band was well defined and of a rather even breadth all the way across the section. In the corium, the subpapillary layer had been replaced by a richly cellular connective tissue, containing apparently some nests or strands of tumor cells. The elastic tissue in this layer had completely disappeared (fig. 15).

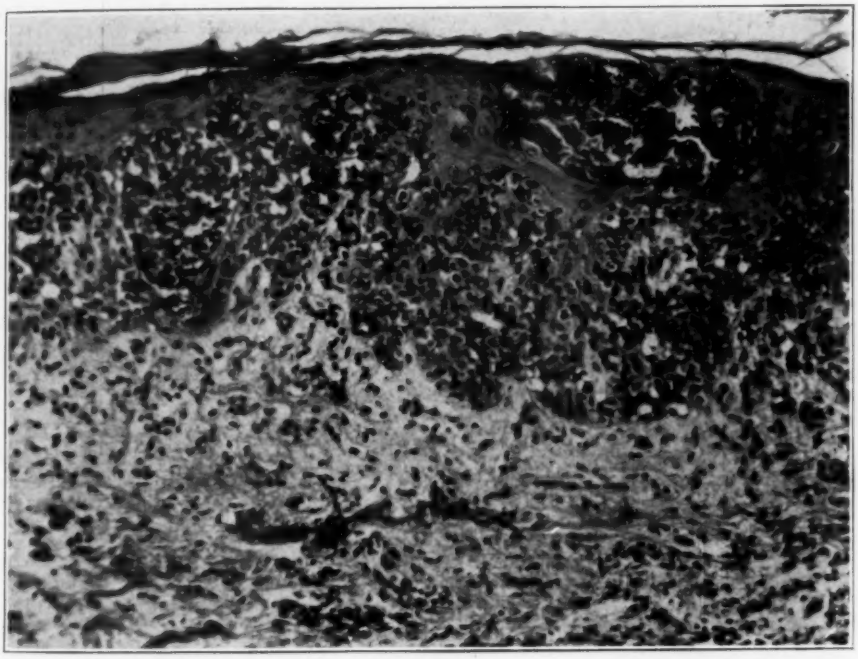

Fig. 11.—Specimen removed for biopsy 3. There is proliferation of cells in the rete pegs and in the papillae.

COMMENT

When this patient was seen for the first time in the clinic, the diagnosis of mycosis fungoides was concurred in by all the members of the dermatologic staff who saw him. A short time afterward he was presented to the Baltimore-Washington Dermatological Society where the same diagnosis was agreed on with no dissension. The absence of itching was the only unusual feature noted, but, according to Ormsby in his book, "Diseases of the Skin," this may occur in typical cases of the condition.

After an examination of the specimen obtained at the first biopsy, however, it was evident that we were dealing with a histologic picture different from that which is usually found in this disease. While the histologic descriptions from various sources have varied somewhat, there has been a greater diversity of opinion as to the nature of the pathologic process. Although probably the majority of investigators at present feel that the histology resembles most that of the infectious granulomas,

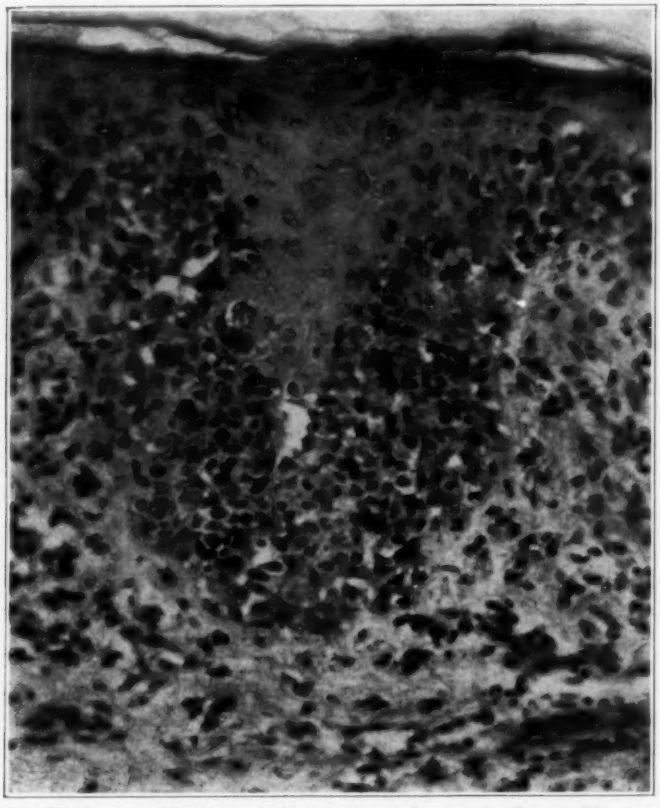

Fig. 12.—High magnification of the same section as in figure 11, showing the solid down growth of a rete peg.

numerous other interpretations as to the nature of the disease have been offered. The most common clinical signs consist [1] of only cutaneous lesions without visceral manifestations or alterations in the blood picture and in most instances even without any evident involvement of the lymph gland. From the histologic standpoint, there is a polymorphous cellular infiltrate, the predominating cell of which appears to be a large

1. Bazin, A. P. E.: Leçons théoriques et cliniques sur les affections cutanées artificielles, Paris, 1862, p. 372.

monocyte of the lymphoid type together with fewer small cells which are probably derivatives of these or of the same series. There are also a variable number of polymorphonuclear leukocytes, plasma cells and a few mast cells and eosinophils.[2] Frieboes [3] considered eosinophils to be characteristic of all stages of the disease. The conception that mycosis fungoides is a lymphadenomatosis is attributable to pioneer

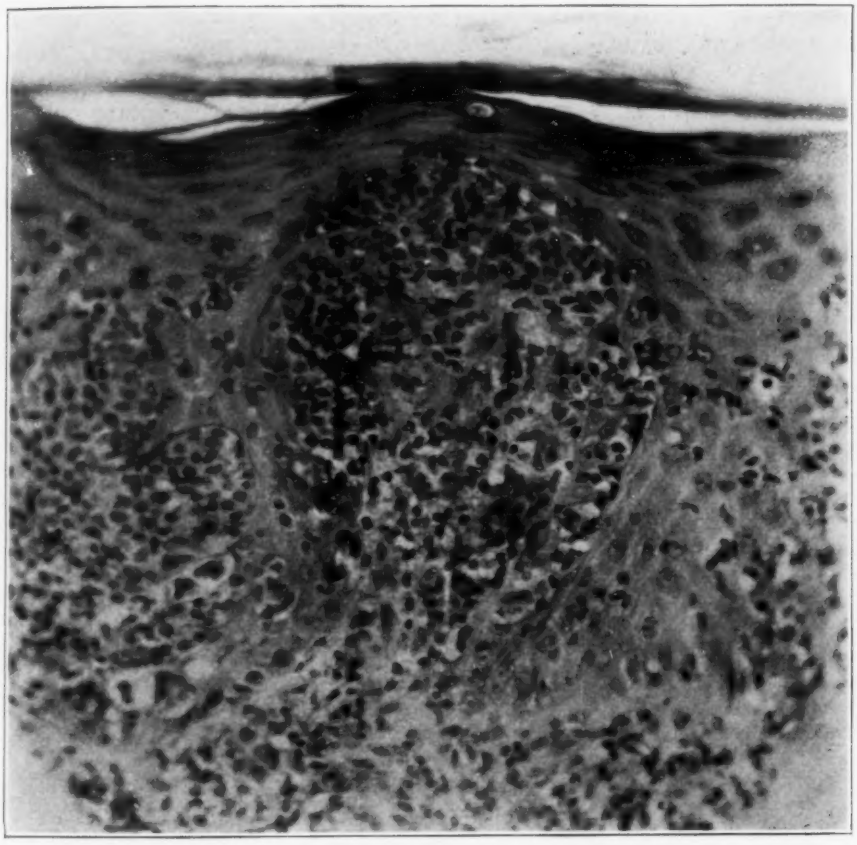

Fig. 13.—High magnification of the same section as in figure 11, showing masses of cells growing into the rete on the left and a large mass in the center, apparently lying in a distended papilla but invading the surrounding epidermis. Here the cells look more like inflammatory cells than those in figure 12.

2. Kyrle, J.: Histo-Biologie der menschlichen Haut und ihrer Erkrankungen, Berlin, Julius Springer, 1927, vol. 2, p. 274.

3. Frieboes, W.: Grundriss der Histopathologie der Hautkrankheiten, Leipzig, F. C. W. Vogel, 1924, vol. 2, p. 222.

investigators [4] of the subject as well as to more modern authors.[5] This can be interpreted as linking up the disease with the leukemias and aleukemias. A great number of authors [6] have adopted the view that the disease is an infectious granuloma because of the polymorphous character of the cellular infiltrate in the cutaneous nodules and also in the nodules of internal organs in such cases as have shown visceral involvement. Duhring,[7] impressed by the early inflammatory symptoms and later by the developments of a sarcomatous aspect, referred to the condition as inflammatory fungoid neoplasm. Other investigators [8] have concluded also that it may be a form of sarcomatosis. An occasional report in the literature has emphasized a specific histologic picture with the conclusion that the disease constitutes a separate entity.[9] Symmers [10] believed that mycosis fungoides fell into an as yet unnamed group of diseases which includes Hodgkin's disease, Cohnheim's pseudoleukemia and related conditions. His interpretation of Hodgkin's disease probably revealed his point of view with regard to this entire subject. He felt that this diease was neither a pure granuloma nor a neoplasm, but that on the one hand it partook of the nature of an infectious granuloma and

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4. Gillot, Xavier: Étude sur une affection de la peau décrite sous le nom de mycosis fungoïde (lymphadénie cutanée), Thèse de Paris, 1869, no. 85. Cornil, V., and Ranvier, L.: Manuel d'histologie pathologique, Paris, Félix Alcan, 1901, vol. 1, p. 430.

5. Pardee, L. C., and Zeit, F. R.: Mycosis Fungoides, J. Cutan. Dis. **29**:7, 1911. Strobel, E. R., and Hazen, H. H.: Mycosis Fungoides in the Negro, ibid. **29**:147, 1911.

6. Köbner, H.: Histologisches und Bacteriologisches über Mycosis fungoides (Alibert), Fortschr. d. Med. **4**:549, 1886. Stelwagon, H. W., and Hatch, J. L.: A Study of Mycosis Fungoides with a Report of Two Cases, J. Cutan. Dis. **10**:1 and 51, 1892. Galloway, J., and MaCleod, J. M. H.: Mycosis Fungoides, Brit. J. Dermat. **12**:153, 1900. Paltauf, R., and von Zumbusch, L.: Mycosis fungoides der Haut und inneren Organe, Arch. f. Dermat. u. Syph. **118**:699, 1914. Auspitz, H.: Ein Fall von Granuloma fungoides, Vrtljsschr. f. Dermat. u. Syph. **12**:123, 1885. Payne, J. F.: Granuloma Fungoides, Tr. Path. Soc., London **27**:22, 1886.

7. Duhring, L. A.: A Practical Treatise on Diseases of the Skin, ed. 3, Philadelphia, J. B. Lippincott Company, 1888, p. 560.

8. Kaposi, M.: Pathology and Treatment of Diseases of the Skin, New York, William Wood & Company, 1895, p. 593. Bowen, J. T.: Mycosis Fungoides and Sarcomatosis, J. Cutan. Dis. **15**:65, 1897. Mackay, H., and Boyd, W.: A Contribution to the Pathology of Mycosis Fungoides, J. Cutan. Dis. **36**:521, 1918. Ormsby, O. S.: Diseases of the Skin, Philadelphia, Lea & Febiger, 1915, p. 824. Goetheil: Discussion, J. Cutan. Dis. **29**:16, 1911.

9. Knowles, F. C.: The Histopathology of Mycosis Fungoides, J. Cutan. Dis. **33**:563, 1915.

10. Symmers, D.: A New Interpretation of the Pathologic Histology of Hodgkin's Disease, Arch. Int. Med. **19**:990 (June) 1917.

on the other of a systemic lymphosarcomatosis. Vidal and Brocq [11] and Breakey [12] found lymphosarcoma in their cases, and in recent reports based on numerous cases there seems to be a tendency by some to place the disease in this category. Fraser [13] found pathologic changes that were typical of lymphosarcoma and decided that the lesion of mycosis fungoides had its origin in the reticulum cells of the papillary layer, that is, the reticulo-endothelial system of the skin. Keim [14] found the predominant cellular structure to be composed of round cells of the lymphocytic type in varying stages of differentiation. He placed the disease

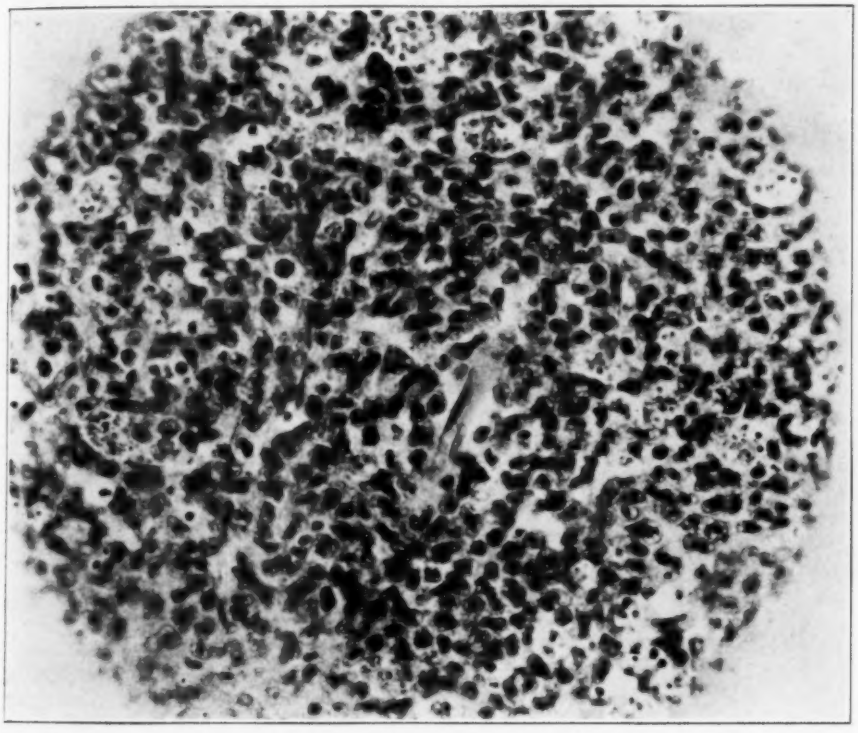

Fig. 14.—High magnification of a fully developed tumor from the specimen removed for biopsy 7. Note the marked cellular fragmentation and mitosis.

11. Vidal and Brocq: France méd. **2**:946, 1885.

12. Breakey, W. F.: A Case of Mycosis Fungoides, J. Cutan. Dis. **20**:316, 1902.

13. Fraser, J. F.: The Pathology of Mycosis Fungoides, J. Cutan. Dis. **35**:793, 1917; The Interpretation of Mycosis Fungoides as a Variety of Lymphosarcoma, Arch. Dermat. & Syph. **11**:425 (April) 1925; Mycosis Fungoides: Its Relation to Leukemia and Lymphosarcoma, ibid. **12**:814 (Dec.) 1925.

14. Keim, H. L.: The Lymphoblastomas: Their Interrelationships, Arch. Dermat. & Syph. **19**:533 (April) 1929.

in the group of true lymphadenotic infiltration of the skin in which he included also leukemia, lymphosarcoma, Hodgkin's disease, Sternberg's leukosarcoma and Kaposi's lymphoderma perniciosa. He felt that the multiple clinical pictures associated with these diseases should be regarded as the variable cutaneous expressions of the lymphoblastomas.

In our survey of the literature on mycosis fungoides, we have been able to find only one report of a suspected case in which the pathologic process suggested an epithelial origin. This case, recorded by Schmidt,[15]

Fig. 15.—Specimen removed for biopsy 8 showing the stage of resolution with the formation of new connective tissue in the upper layers of the corium and absence of elastic tissue. Groups of tumor cells are seen in the epidermis.

resembled clinically mycosis fungoides d'emblée, but the author concluded that the lesions were true tumors, most probably a peculiar type of carcinomatosis cutis.

In the study of our case we were convinced early that the pathologic process had its origin in the epidermal structure. In order more thor-

15. Schmidt, F. R.: Multiple Tumors of the Skin Resembling Mycosis Fungoides, Arch. Dermat. & Syph. **18**:231 (Aug.) 1928.

oughly to establish its noninflammatory character, Dr. Arnold Rich made some supravital stains with neutral red, which showed a practical absence of phagocytic cells. In the early stages, however, such as is shown in figure 6, it was not possible to feel certain that all of the cells lying around the epithelial pegs came from epithelium. However, their arrangement and almost complete confinement to the basal layer as seen in figure 5 certainly suggested it. But as the process extended and the rows of cells became wider, filling in the papillae, it is quite likely that some of the cellular elements were not of epithelial origin. This possibility becomes less remote when one considers that the earliest change found in an erythematous lesion was a mild inflammatory reaction in the corium. In any case, none of the cells were the easily recognized inflammatory types, such as polymorphonuclear leukocytes and plasma cells.

The most interesting phase in the pathogenesis of the process, as suggested by our material, was the apparent initiation of the process in the epidermis by cell death or at least damage, as shown by the pyknotic nuclei, rather than by malignant proliferation. To be sure, the latter process soon became evident, especially in small groups of cells in the rete which had become disorganized and showed mitotic figures. Cellular fragmentation was a striking feature, appearing early, even in the smallest collections of cells that showed evidence of malignant proliferation.

In considering the question as to what could cause such a widespread tendency for a malignant proliferation of the epidermal structures, we have thought that there might be a circulating agent which had disturbed the normal balance between the dermal and epidermal layers. With elastic tissue and connective tissue stains, including Foot's modification of Bielschowsky's method, however, we were unable to demonstrate any abnormalities in the dermo-epidermal border. This, of course, does not exclude such a possibility.

Another interesting feature of the pathologic process in this case was the apparent simultaneous involvement of the epidermis and the epidermal appendages. In the specimen obtained for biopsy 2, as well as other lesions, portions of hair follicles, sweat glands and sebaceous glands were found to be affected, which were in such locations as to preclude any probability of direct extension of the tumor cells from the epidermis, although these cells showed a very early tendency to wander down into the connective tissue.

In our attempt to delineate the various histologic stages of the pathologic process, we, of course, do not wish to imply that such a sequence of events in its entirety was followed by all of the cutaneous manifestations. Certainly a stage of resolution is not likely to occur always, and when it does take place, it may follow any of the various types of cuta-

neous lesions. The pathologic process in the connective tissue afterward would depend on the amount of damage that had been done by the malignant cellular invasion.

CONCLUSION

1. We have reported a case clinically resembling mycosis fungoides that showed mixed cutaneous lesions, varying from areas of simple erythema to tumors.

2. The pathologic process was caused, however, by a malignant proliferation of the epidermal structures that did not resemble the usual or any of the variable histologic changes that have been reported in this disease.

ADDENDUM

The patient came to Baltimore again on June 1, 1931, almost one year after the foregoing report was written. In this time he had made five or six visits to another city where he had received roentgen treatments. There had been a marked decrease in the number of lesions, especially the urticarial and erythematous types. Most of the lesions over the buttocks, seen in figure 3, had disappeared without roentgen therapy. There were three or four ulcerating nodules here and there and a raised, foul smelling, fungating tumor, 4 or 5 cm. in diameter, on the left thigh which had had some radiation but had not as yet shown any evidence of healing. There were also two or three nodular lesions, 1 or 2 cm. in diameter, on the thighs, which had not ulcerated, and here and there there were some infiltrated plaques of various configurations, varying in size. The large tumor on the left arm, shown in figure 4, had entirely disappeared leaving only a slight scar with partial loss of hair in the center. It had received 1.5 skin units of roentgen rays just before the patient left Baltimore. The back and the chest were practically free from lesions, and many of those that were present on the upper portion of the arms one year before had disappeared. The patient had continued to feel quite well. There were no enlarged glands except in the inguinal regions which might well be accounted for by a secondary infection of the ulcers on his legs. His blood was studied carefully for evidence of any unusual pathologic cells, but nothing abnormal was found except an increase in the granulocytes. There were 5 per cent monocytes.

Six additional biopsies were made, principally to determine more extensively than had been done before whether or not the tumor cells would store vital dyes. Two lesions were injected with 1 per cent trypan blue, two with 1 per cent saccharated iron oxide and two with Higgins' india ink. The vital dyes were injected into the lesions with a very fine needle, and as superficially as possible. The biopsies were made from one to four days after the stains were injected.

Histologic study showed that only a very few cells had taken the trypan blue stain. With the saccharated iron oxide and india ink many more cells were stained, but these were apparently mostly limited to areas where the stain in concentrated form had come in direct contact with them. There was apparently no selective tendency whatever for the tumor cells to take up the injected material.

ABSTRACT OF DISCUSSION ON PAPER BY DR. ELLER AND DR. FOX AND ON THAT BY DR. KETRON AND DR. GOODMAN

DR. J. FRANK FRASER, New York: I wish first to talk about the case reported by Dr. Eller and Dr. Fox. There may be room for doubt about the clinical diagnosis, but there can be no question concerning the nature of the pathologic changes. The histologic picture reported here is exactly the same as that seen in the tumor stage of mycosis fungoides. In 1925, I suggested (ARCH. DERMAT. & SYPH. **12**:814 [Dec.] 1925) that the lesion of mycosis fungoides had its origin in the reticulum cells of the skin, in other words, the reticulo-endothelial system of Aschoff or the adventitial mesenchymal cells described by Marchand. These are the cells that dominate the picture in this case. The lesion should not be classified as an inflammatory process because many of the essential histologic features are those of neoplasm.

The neoplastic features are: (1) uniformity of the cells; (2) numerous mitoses, and (3) hyperchromatism of the nuclei.

Two important questions are suggested:

1. What evidence is there from the reported cases of iododerma that iodine can produce these changes in the reticular cells? Apparently there is none. In the cases collected by Dr. Eller and Dr. Fox the histology was reported in only one, and that picture bore no resemblance to the microscopic observations in the present case.

The second closely associated question is:

Have we any evidence from the reported cases of mycosis fungoides that iodism played any part as an etiologic factor? To my knowledge there is no such evidence. It might be well to develop some experimental technic with the view of finding a solution of these questions.

Speaking of Dr. Ketron and Dr. Goodman's case, there can scarcely be any disagreement in regard to the clinical features; they all point, except for the absence of itching, to the diagnosis of mycosis fungoides. Any difference of opinion must arise in regard to the interpretation of the histologic picture. The authors have interpreted the pathologic process as a "malignant proliferation of epidermal epithelium."

The main question to be decided is whether the cells in these lesions are a proliferation of epidermal cells or of foreign cells that invade the epiderm. The histologic picture brings to mind very strongly the picture seen in Paget's disease of the nipple (fig. 7). The traditional discussion as to the origin of the cells in Paget's disease shows how difficult it is to decide whether the pathologic cells are invaders or a proliferation of epidermal cells in situ. There are those who say that the Paget cells are changed epidermal cells and others who are equally certain that they are an invasion from the ducts. Most of the histologic pictures seem to favor equally both views.

One point developed in the paper by Dr. Ketron and Dr. Goodman, which appears to support strongly the theory of the epidermal origin of the lesion, is what is claimed to be a demonstration by MacCallum's bacterial stain of intercellular bridges between the tumor cells. One should make certain that the bridges that appear in these sections do not belong to epidermal cells that have been invaded by the new cells, rather than to the new cells themselves. In other words, a tumor cell may invade the epidermis, and when it is lying between two or more epidermal cells one may see bridges of cytoplasm from the surrounding epidermal cells radiating toward the nuclei of the tumor cells. This appearance is especially evident when the cytoplasm of the new cells is vacuolated and degenerated. If the bridges belonged to the tumor cells they ought to be present in the cell nests of the corium. We must remember that the reticular processes of the reticulo-endothelial cells extending from the cytoplasm of one cell to another often may present the appearance of intercellular bridges.

The second fact claimed by the authors in favor of the epithelial origin of the cells was the absence of phagocytic cells as shown by supravital stains. The phagocytic character of the reticulo-endothelial cells is not always shown even when these cells are normal, and we should not expect to find it in neoplasms, as the energy of function in neoplastic cells is largely suppressed in favor of that of growth.

A strong point it seems to me against the epithelial origin of these lesions is the total dissimilarity of the histologic picture to that of the picture we see in those cases of carcinoma that are undoubtedly derived from epidermal epithelium. I mean the common cancers of the skin with which we are so familiar.

My personal view of the case is that there is sufficient evidence from the clinical and histologic features to justify the diagnosis of mycosis fungoides.

DR. SIGMUND POLLITZER, New York: The two papers to which we have just listened present features of extraordinary interest and importance beyond the field of cutaneous pathology with which they deal.

In regard to the case of iododerma reported by Dr. Eller and Dr. Fox, obviously the first question to be considered is, what are we dealing with clinically in this case? I do not see how there can be more than one answer to this question. Cases of bullous, nodular and tuberous lesions resulting from the prolonged ingestion of iodides have been recognized and reported in the literature for more than half a century. The case in question, which, through the courtesy of Dr. Eller I had an opportunity of examining, impressed me at my first examination as bearing a striking resemblance to Sir Jonathan Hutchinson's case of fatal iododerma. The aspect and course of the lesions, in connection with the established fact that the patient had been ingesting small quantities of iodide daily over a period of several years and therapeutic doses for several weeks before his eruption appeared and was voiding considerable quantities of iodides through the kidneys for several months after he had ceased taking iodide, and that chemical examination of many tissues and organs post mortem showed that he had retained large quantities of the drug left no room for doubt as to the cause of his disease. That literally millions of people are regularly taking minute doses of iodide in the form of iodized table salt, and that countless numbers of people have ingested large doses of iodide for long periods without developing a cutaneous disorder has no more significance than that of the millions of people who have received repeated doses of arsphenamine only a very small number have reacted with an arsphenamine dermatitis. In all such cases, it can hardly be necessary to say, there is an unknown factor that makes the special reaction possible, but the arsphenamine or the iodide is a neces-

sary link in the causal chain that results in the reaction that we properly term arsphenamine dermatitis or iododerma, respectively.

The histologic picture in this case presented such difficulties of interpretation that competent pathologists who examined the sections made diagnoses such as a pigmentary melanotic carcinoma, lymphosarcoma, metastatic carcinoma probably from adrenal rests, etc. I need not repeat what the reader of the paper has already stated. The process as first pointed out by Dr. Paul Klemperer seemed to originate in the adventitial cells of the smallest vessels in the cutis and to be a proliferation of these mesenchymal cells. This adventitial cell growth ultimately fills the entire cutis at the affected regions, extends to the papillary layer and causes an obstructive edema of the epidermis with a breaking up of the epidermic layer and to some extent a reactive epidermic proliferation. Etiologically and histologically, the case is sufficiently clear.

Now to consider Dr. Ketron and Dr. Goodman's case of a peculiar epitheliomatosis of the skin. Clinically, the case seemed so obviously a mycosis fungoides that no one of the many dermatologists who saw the patient suggested any other diagnosis, and yet, it must be admitted that certain features that belong to the clinical picture of mycosis fungoides were missing. There was nothing of the usual prefungoid erythema, and there were none of the prominent fungating tumors that form so striking a feature of mycosis fungoides. When a histologic study of their case revealed peculiarities in the epidermis, the authors, regarding these as the dominant feature, came to look on the case as a peculiar form of multiple carcinosis of the skin. Now, it is a cardinal principle in pathology that the histologic picture must conform to and explain the clinical picture. Does it seem reasonable that this process going on now for more than three years, consisting of literally hundreds of ulcerating lesions many of which heal and disappear without leaving a scar, can be a cancerous process? Such a view contravenes all our knowledge of the pathology of cancer.

I am indebted to Dr. Ketron for an opportunity to examine a number of his slides. The picture to my mind was that of a reticular cell tumor. The histology of mycosis fungoides has for many years been a subject of controversy among pathologists. Without entering into a discussion of the various views, I wish to range myself emphatically with Dr. J. Frank Fraser (ARCH. DERMAT. & SYPH. **12**:816 [Dec.] 1925), who was the first to state clearly that the cells making up a mycosis tumor are derived from the adventitial cells of the small blood vessels of the skin, a view that has been corroborated (unfortunately, without acknowledgments to Fraser) in a paper by Berger and Vallée (*Presse méd.* **38**:117 [Feb. 5] 1930) published five years later.

To digress for a moment, three years ago, Schmidt (ARCH. DERMAT. & SYPH. **18**:231 [Aug.] 1928), of Chicago, published a case in many respects like that of Dr. Ketron and Dr. Goodman. He regarded the condition as mycosis fungoides, but on the basis of the histologic picture was led to accept Sternberg's (Vienna) interpretation that it was a multiple carcinosis of the skin. The same patient later came under the care of Beeson and Hueper, of Chicago, who made further histologic studies on fresh and autopsy material (ARCH. DERMAT. & SYPH. **19**:794 [May] 1929). Their conclusion was that the case was one of multiple sarcomatosis of the skin. I mention this case to illustrate the divergence of opinions on the part of the ablest pathologists when it comes to the interpretation of mesenchymal tumors of the skin. The same contradictory diagnosis was made in the case of Dr. Eller and Dr. Fox. All these difficulties vanish when the reticuloendothelial cells of the skin are taken into consideration, and it is remembered that these cells have the polyvalent potentialities of embryonal cells.

In Dr. Ketron and Dr. Goodman's case, I looked carefully for evidences of epitheliomatous proliferation in the epithelial layer over the lesions. I was unable to find anything that I could interpret as cancerous. The epithelial layer was subject to great stress from the edema and pressure of the new tissue, its continuity was broken and the new cells encroached on and to some extent penetrated into the epithelial layer. Such changes are not infrequent in the case of neoplasms in the cutis and even in acute inflammatory processes. These changes are not cancerous. The cells in the cutis are mesenchymal, not epithelial, and the changes in the epidermis are secondary.

How then shall we interpret Dr. Ketron and Dr. Goodman's case? Both mycosis fungoides and iododerma are examples of reticulo-endotheliosis of the skin. Both patients in question were victims to the prolonged ingestion of iodide. Can we differentiate between the reticular cell proliferation of mycosis fungoides and that produced by iodide? There is only one feature in the histologic picture of mycosis fungoides that may be sufficiently characteristic to enable us to make a diagnosis: In the tumor stage of mycosis fungoides there is an extraordinarily rapid development and destruction of the new cells so that nuclear fragmentation (karyorrhexis) is a prominent feature in the disappearing mycosis tumors. In the case of Dr. Ketron and Dr. Goodman there was a notable amount of what the authors called chromatin dust scattered throughout the field. I am not sure that this feature is enough to establish a diagnosis of mycosis fungoides, though I probably should have made that diagnosis if the patient had not, like Dr. Eller and Dr. Fox's patient, been a chronic iodide addict. In short, I am in doubt as to the pathogenesis of the case of Dr. Ketron and Dr. Goodman, but I incline to the view that it is a replica of Dr. Eller and Dr. Fox's case.

Whether the former is a case of mycosis fungoides or iododerma, the great importance lies in the circumstance that we have in these two conditions an example of similar effects produced by different causes. The reticulo-endotheliosis of iododerma has the prolonged effect of iodide as its dominant factor; the reticulo-endotheliosis of mycosis fungoides is due to causes wholly unknown. A valuable secondary effect from these presentations may be expected in the focusing of attention on the reticulo-endothelial system and the clearing up of some obscure problems in the pathology of the skin.

Dr. Udo J. Wile, Ann Arbor, Mich.: Like Dr. Fraser and Dr. Pollitzer, I had the opportunity of seeing both of the patients, and also, through the kindness of Dr. Eller and Dr. Ketron, of studying the slides from these two most unusual cases.

In regard to the case of Dr. Eller and Dr. Fox, I find myself entirely in accord with Dr. Fraser's view, and I disagree with the views expressed by Dr. Pollitzer. I do not feel that the finding of iodine in the patient who has ingested iodized salt is more than suggestive evidence of the etiology of his eruption, if that eruption is not typical of the usual drug exanthem, and I should also like to direct attention to the fact that if this particular disease picture, which certainly histologically resembled mycosis fungoides, was caused by iodine, that in this particular case iodine behaved entirely differently than when ingested under ordinary conditions. Iodine passes with the greatest ease through the kidney tubules. It has practically no tendency to be stored up in the tissues. It passes through the renal epithelium so readily that it seems inconceivable that if a patient has an eruption so severe as this patient had when I saw him, with no iodine in the urine for a month, that iodine could have been a factor in establishing this extraordinary manifestation.

Another point, to which exception may be taken, is in warning the medical profession against the indiscriminate use of iodized salt; I do not feel that is at all a justifiable conclusion. We who live in the goiter belt use iodized salt almost exclusively, in some instances by law and in some because of rules of the state board of health. I dare say that little other than iodized salt is used in the entire state of Michigan and in the entire goiter belt. Even if this case were iododerma as the result of iodized salt, I do not think the medical profession need be warned against the use of iodized salt.

With regard to Dr. Ketron's case, I have had an opportunity of studying a large number of cases of mycosis fungoides with the late Dr. Warthin. I had an opportunity to see the slides before I saw the patient, in this instance. Clinically, the condition was mycosis fungoides. The changes in the epithelium, I thought, were entirely like those seen after irradiation of the skin, particularly in mycosis fungoides, and I raise the question whether these changes were not those of degenerative type rather than proliferative, a change one commonly sees following irradiation.

I find myself entirely in accord with Dr. Fraser's view that these two cases are mycosis fungoides. It is entirely possible, as stated by Dr. Eller and Dr. Fox, that a retention of iodine in the blood may provoke such a change, but nevertheless I do not think that in their case we are dealing with a true iododerma. I am very sorry that the bone marrow and lymph glands were not studied, for I think they might have shown changes that would have thrown light on the lymphoblastomatous nature of the process.

Dr. W. H. Goeckerman, Rochester, Minn.: This case reminds me of one of our cases, the report of which is to be published. In our case bullous lesions were a striking part of the eruption, and we made a clinical diagnosis of the bullous type of mycosis fungoides, which is seemingly a rare clinical picture. The similarity to this case was particularly emphasized by the apparent epithelial origin of the lesions. So true was this that two competent pathologists decided we were dealing with unusual epithelioma possibly akin to Paget's disease. Sections were submitted to a number of pathologists in different parts of the country, and the predominating opinion was in favor of an unusual lymphoblastoma. Dr. Montgomery and I, who are reporting the case, felt that it furnished contributory evidence for the opinion that the various lymphoblastomas are different clinical manifestations of genetically the same disease.

Dr. Fred Wise, New York: I also had the opportunity of watching the patient of Dr. Eller and Dr. Fox, and noting the changes in the eruption almost from the beginning to the end. In its early stages there was no difficulty in identifying the eruption as one due to iodides, especially with respect to the tuberous lesions on the face. But as the disease progressed and severe constitutional symptoms set in, the picture changed to one of extreme malignancy, so that one was reminded of a generalized "galloping sarcomatosis." The lesions assumed the characters of a rapidly advancing, fungating mycosis fungoides in some areas, and of a rupioid syphilid in others. The fact that the disease progressed long after the ingestion of iodides was stopped tended to cast doubts on the original diagnosis, and the uncertainty was greatly increased when the histopathology came under the consideration of a group of pathologists. I have seen several instances analogous to this one, except that the patients recovered, from the ingestion of bromides. One of these I saw last year in Bloch's clinic in

Zurich, the case of a woman whose body was almost covered with the nodular and fungating tumors of bromoderma. I believe that fresh lesions of bromoderma may manifest themselves long after the drug has been stopped, and I have seen a few instances in which the same phenomenon might take place with respect to phenolphthalein, although of this I am not sure.

In the case of Dr. Eller and Dr. Fox, the fact that complete involution of many lesions took place within a few days, leaving an apparently normal skin in situ, eliminated, to my mind, the question of malignant neoplasms, in the sense of cancer or sarcoma.

Dr. Clark W. Finnerud, Chicago: I have not seen the sections, and my opinion is based entirely on the illustrations.

In spite of the interesting points of similarity between the cases in these two papers I shall limit my discussion to Dr. Ketron and Dr. Goodman's presentation.

Having had a somewhat similar histologic picture to contend with at one time, I should like to ask Dr. Ketron if any differential stains other than those mentioned were employed. In view of the illustrations and the description presented, I should not consider this an epithelial proliferation. It is sometimes difficult clearly to distinguish proliferating and infiltrating cells of the papillary corium from epidermal structures, particularly in the presence of dyskeratosis and rather dense cellular infiltration.

The diagnosis is, of course, merely a matter of interpretation, as stated by Dr. Fraser. The epidermal changes to me appear to be nutritional. The cells considered basal cells in the early lesions do not appear to me to be of that type.

Tentatively, rather than to attempt to make an exact diagnosis, I should be inclined at least to group this case in a general way with the lymphoblastomas, adenotic infiltrations, to include mycosis fungoides.

Dr. Walter J. Highman, New York: I had no opportunity to study the case of Dr. Eller and Dr. Fox closely, but one night I saw the patient by flashlight. The clinical diagnosis that appeared to me likely to be correct, in spite of the history, was either pemphigus vegetans or mycosis fungoides. I considered the history and discounted iodine as the cause. The only histologic specimen I saw was poor, but it struck me as belonging among the lymphoblastomas. I suggested that Dr. Eller consult Dr. Klemperer. My impression was that, whatever the immediate etiology of the lesion, in effect the condition belonged in the group that has been emphasized by both Dr. Fraser and Dr. Pollitzer, and a little bit more tentatively by Dr. Wise.

What seems to me most significant is whether this case may be a key to the understanding of mycosis. Last week in Philadelphia Furth published his work on a filtrable virus, related to leukemia, I believe in poultry. The kinship of leukemia, mycosis and the lymphoblastomas is at least conceded to be a likelihood, and if leukemia can be produced by a filtrable virus, and I do not know why not, I do not know why iodine may not be accountable for a picture that is at least similar to mycosis. In that light, it is conceivable that in this whole group of lymphatic diseases perhaps the thought is not acceptable that they are due to a virus, whatever it may be. I think that this is the significance of the case reported by Dr. Eller and Dr Fox.

What the significance of the case of Dr. Ketron and Dr. Goodman may be I do not know. I am so completely in accord with the previous speakers that I can add nothing in particular, as I do not wish a wrangle about laboratory details. I

feel very much, when we haggle here about such details, like the old lady who asked a keeper in the zoo about the sex of the rhinoceros. The keeper replied that it could interest only another rhinoceros.

DR. HOWARD MORROW, San Francisco: I brought a few photographs for Dr. Ketron, which suggest a condition quite similar to the one which he reported. Clinically, the lesions were sarcoma-like. Some of the pathologists who saw the patient considered the condition a lymphosarcoma, others mycosis fungoides, but we fell back on the old term lymphoblastoma. About that time during my absence Dr. Mook visited San Francisco and saw this patient, and at his suggestion the patient was given some injections of a patented German preparation supposed to contain the element cerium, and to our surprise the eruption entirely disappeared. This is not reported as a cure, but as a peculiar coincidence, which Dr. Ketron may take advantage of.

DR. BEDFORD SHELMIRE, Dallas, Texas: Some three years ago, in an article published in *The Journal of the American Medical Association,* I called attention to acneform eruptions due to the prolonged ingestion of iodized salt. Due to extensive advertising of this salt in my section of the country, many persons, especially women, ingest an appreciable amount of this condiment. I have known many women to ingest a teaspoonful of iodized salt in a glass of warm water on arising each morning. This was done with the hope of "preventing that lump" in the neck.

More serious types of cutaneous eruptions may be seen following the ingestion of this salt. Two widespread pustular eruptions of the face and scalp in men were found to be due to this salt. On removal of the iodized type of table salt from the diet of these persons, the cutaneous eruption disappeared spontaneously but recurred within ten days or two weeks after the use of iodized salt was continued. By this same procedure of therapeutic testing, an iododerma of the legs and an erythema nodosum were found to be due to iodine in the salt.

As some persons develop a cutaneous eruption following ingestion of a few grains of iodine, it is not improbable that iodized salt played an important rôle in the iododerma presented by Dr. Eller.

DR. SAMUEL AYRES, JR., Los Angeles: I merely wish to call attention to one point in the history, which was not followed up. The patient had a blood sugar of 146. My conception of abnormal blood sugar is anything above 120. I think it would have been well to have done a dextrose tolerance test, and the use of insulin might have been worthy of investigation. It is possible that the high blood sugar in conjunction with the iodine may have brought about this peculiar clinical picture. It is unfortunate that this cannot be checked up.

DR. C. GUY LANE, Boston: In connection with the paper of Dr. Eller and Dr. Fox, Dr. C. Morton Smith has just given me this photograph of a patient whom he saw thirty-three years ago, with a fatal iododerma. The patient was seen in 1898, and gave a history of having been taken sick some nine days previously, and of having taken potassium iodide three times a day, apparently for seven days before entering the hospital. The eruption became vesicular, bullous, hemorrhagic and ulcerative, and progressed, and sixteen days after the onset the patient died with a very extensive eruption, as you can see in the photograph.

I was also interested in the recent article by Perutz and Guttmann, who have been discussing the disturbance of sodium chloride and water metabolism in cases of dermatitis herpetiformis. They were apparently able to demonstrate that the iodine sensitivity in some instances could be increased by increasing the supply of sodium chloride, and a decrease in the chloride in the tissues apparently

lessened the iodine sensitivity. They suggested that an increase in the sodium chloride content can perhaps hinder the formation of vesicles. Whether this can be worked out in practice I do not know.

DR. EARL D. OSBORNE, Buffalo: A great deal has been said about the pathologic process in these cases. I believe that something more should be said regarding the chemistry of the iodides. I was interested in one statement which Dr. Wile made about the minuteness of the dose of iodine in iodized salt, and the seeming impossibility of the production of severe iodide lesions from such a small dose of iodine. I firmly disagree with his statement on account of some work I did nine or ten years ago on the pharmacology and therapeutics of iodides which was published in *The Journal of the American Medical Association.*

We found that the production of an acute bullous iododerma had nothing to do with the size of the dosage ingested, but was dependent on the degree of sensitivity of the individual to the iodide. Furthermore, we demonstrated that in sensitive persons, potassium iodide would produce a more severe and extensive eruption than would the same dose of sodium iodide. This observation has been reported in the literature by other observers.

In our chemical studies we employed the method of Kendall for the detection of iodine. This method is the most sensitive known for the detection of this chemical and will detect one part in one millon. We showed that none of the iodine was taken up by the cellular elements of the blood after administration of iodide by mouth or intravenously. The protein fraction of the plasma was then separated from the nonprotein fraction, and it was shown conclusively that, following the administration of potassium iodide, a much higher percentage of the iodine was taken up by the protein fraction than following the administration of sodium iodide. In other words, following the use of potassium iodide, there is a protein potassium iodine combination, whereas following the use of sodium iodide there is a protein sodium iodine combination to a much lesser degree. I recall this work to your attention and offer it as a very probable explanation of the reaction following the ingestion or intravenous administration of the iodides.

The foregoing work offers a rational explanation for the severe anaphylactoid type of shock that occasionally follows the intravenous administration of sodium chloride in the treatment for iododerma. Dr. Wile has previously called attention to the fact that in severe iodism or brominism the sodium chloride should be administered cautiously and beginning in small doses. When the protein sodium iodine combination or protein potassium iodine combination is split up too rapidly, the liberated protein supposedly produces a condition similar to anaphylactoid shock. Clinical experience bears this out.

Further, we know that practically all metals produce their damage to the skin in the vascular network in the subpapillary layer of the corium. I should like to suggest that in the case of Dr. Eller it was the protein potassium iodine foreign body that produced the reaction in his case and other similar cases of iododerma.

Another point is that if one does not find iodine in the urine it is not necessarily a sign that there is no iodine in the body in excess of normal amount. If iodides can form a chemical combination with the proteins in the blood stream there is reason to believe that a certain amount of iodine may be retained in the circulation and not show in the urine. In one very severe case of bullous iododerma studied at the Mayo Clinic we failed to find more than the usual trace of iodine in the urine although we were able to produce a very severe acute iododerma on three occasions with 1 grain (0.065 Gm.) of sodium iodide. This patient was eventually desensitized by starting with very minute doses of sodium iodide and gradually increasing

them. The fact that a patient can be desensitized to iodides suggests a combination between the protein of the blood stream and the iodides.

Dr. Joseph J. Eller, New York: Regarding the importance of iodine in the urine, Dr. Osborne explained that very well. Apparently it was stored up in this patient, for during the first few weeks the patient was under observation iodine was not found in the urine. Then the patient passed iodine daily in large amounts, which was here mentioned. Necropsy showed 0.2 Gm. of iodine to 100 mg. of the skin, and a large amount in the kidneys and liver.

I think that the discussers of this paper have covered the ground very thoroughly.

Dr. Lloyd W. Ketron, Baltimore: We have presented this case with a certain amount of trepidation because we had warnings beforehand that our interpretation of the pathologic process would not go unchallenged. However, after a study of many biopsy specimens taken over a period of many months, we felt that our view was the correct one. We are not entirely without support. Dr. Arnold Rich went over the material with us on many occasions, and he was thoroughly convinced that the lesions were of epithelial origin. Dr. W. G. MacCallum inclined toward the same view, but he would not definitely commit himself.

With reference to some of Dr. Fraser's remarks, we did not mean to infer that the epithelial bridges were formed by proliferation of new cells but in the section in which these bridges were stained so well the early proliferation of the basal cells was more impressive than in the other preparations.

The only criterion that identifies the reticulo-endothelial system is the property of its cells to take up and store colloidal vital dyes, and we feel that we have shown quite conclusively that the cells in our case do not show this property. If one wishes to say that they originate from the undifferentiated mesenchymal cells such a claim could certainly be properly made, but as yet it has not been proved that the reticulo-endothelium is derived from these cells.

I feel that Dr. Pollitzer is held down a little too much by tradition in using as an argument against the epithelial origin of this disease the fact that the condition clinically does not conform to our usual varieties of cancer of the skin. It is quite possible that the story as yet is not complete so far as malignant growth of epithelial tissue is concerned.

Dr. Finnerud asked about the stains we have made. We have stained material from about fifteen specimens taken for biopsy with most of the preparations that we felt could be of any value in classifying the type of cell.

In deference to the adverse discussion of my colleagues, whose opinions I value most highly, I, personally, could be persuaded to modify my original contention only to this extent: It is possible that the primary reaction may be of an inflammatory nature, but this reaction has disturbed the dermo-epithelial balance to such a degree that a peculiar malignant response on the part of the epithelium has followed, resulting in tumor formation.

LUPUS ERYTHEMATOSUS DISSEMINATUS*

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Disseminate erythematous lupus was first recognized as a definite clinical syndrome by Kaposi in 1872. Since that time, many cases have been reported and the etiology has been discussed from a number of standpoints. The vast majority of the victims are women. The prognosis is always grave. Over 80 per cent of the undoubted cases of disseminate erythematous lupus that have been reported have resulted fatally within five years. Kaposi originally reported a mortality of 50 per cent.

In recent years this disease has been studied by Goeckerman [1] and by Keefer and Felty.[2] Most authorities agree that the constitutional symptoms vary markedly. There is a lack of physical endurance years before the cutaneous lesions appear. There are irritability and sensitiveness out of proportion to the apparent physical ailments. Vague, sometimes severe, abdominal complaints are noted. Aches and pains in various joints are common. Moderate to marked febrile reactions are usual.

The cutaneous lesions frequently resemble the erythema multiforme group. They have a tendency to become more widespread in the skin than the discoid type of erythematous lupus, frequently resembling patches of dermatitis or erythematous plaques, occasionally bullous. They may appear on the extremities and on the mucous membranes. The chronic type may be present for years, and then, especially in young persons, assume the characteristics of the disseminate form. The diagnosis is based on the protean manifestations. When the cutaneous manifestations predominate, seborrheic or eczematoid derma-

* Submitted for publication, Feb. 15, 1931.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

* Studies, observations and reports from the Dermatological Departments of the Barnard Free Skin and Cancer Hospital and the School of Medicine, Washington University, St. Louis, service of Dr. M. F. Engman and Dr. W. H. Mook.

1. Goeckerman, W. H.: Lupus Erythematosus as a Systemic Disease, J. A. M. A. **80**:542 (Feb. 24) 1923.

2. Keefer and Felty: Bull. Johns Hopkins Hosp. **35**:294, 1924.

titis, erythema multiforme, dermatitis venenata, sarcoid of Boeck, lichen planus, erysipelas or pellagra may be more or less closely simulated. When abdominal symptoms are present, marked pain and diarrhea may suggest cholecystitis, gastric or duodenal ulcer, chronic appendicitis, colitis, etc. If the toxic symptoms are pronounced, typhoid fever, pneumonia, pyemia, rheumatism or malignant endocarditis is considered. Frequent leukopenia makes one suspect typhoid fever.

Goeckerman's study demonstrated that tuberculous adenitis is strikingly prevalent. While he stated that it may not be demonstrated clinically, the retroperitoneal, pelvic and mesenteric groups may be severely affected, as was shown in two autopsies at the Mayo Clinic. This observation had previously been reported by others, including Brooks, Roberts, Reitmann and Zumbusch, Low, Logan and Rutherford, and Sequeira and Balean.

Extreme sensitiveness of these patients to mild injuries or irritants has been known for a long time. Ravogli [3] reported death in two cases subsequent to the injection of 0.001 mg. of tuberculin. Goeckerman expressed the opinion that tuberculin should never be used in the diagnosis or treatment in these cases. Gennerich [4] stated the belief that there is a sensitizing agent produced in the destruction of the lymph glands by an unknown disease, and that a hematogenous distribution of this toxin causes the characteristic lesions.

Goeckerman's opinion seems to be that lymphatic tuberculosis is a fundamental predisposing cause and is not necessarily clinically recognizable. His study suggested to him that patients with disseminate lupus erythematosus are under a cross-fire from tuberculosis and septic infection, the one creating an allergic background and the other periodically exciting explosions in the predisposed and unstable organism.

According to Keefer and Felty, the consensus is against the conception that this disease is tuberculous in origin. They seem to be inclined to agree with MacLeod,[5] who stated that it is not a pathologic entity due to some specific cause, but rather a cutaneous symptom of a peculiar type which may be called forth by a variety of toxic or septic causes. MacLeod linked the disease with the great group of the "toxic erythemas," and called attention to the observation of Libman and Sacks,[6] who reported four cases of an unusual type of endocarditis in two of which there were lesions closely resembling those of acute lupus erythematosus together with other erythematous eruptions.

3. Ravogli: J. Cutan. Dis. **33**:266, 1915.

4. Gennerich: Arch. f. Dermat. u. Syph. **135**:184, 1921.

5. MacLeod, J. M. H.: Lupus Erythematosus, Arch. Dermat. & Syph. **9**:1 (Jan.) 1924.

6. Libman and Sacks: Tr. A. Am. Physicians **38**:46, 1923.

Keefer and Felty summarized their study by stating that in all probability lupus erythematosus disseminatus is a clinical entity, nearly always occurring in females and characterized by: fever; pain in the muscles and joints; pain and swelling of the joints, but no arthritic changes; abdominal pain; glandular enlargement, sometimes associated with splenomegaly; typical disseminated lesions of the skin; great prostration, occasionally with delirium; secondary anemia, often with a normal leukocyte count; a downhill course with increasing asthenia and failure, and finally death due to an intercurrent infection of the respiratory tract or to nephritis. In its clinical aspects, the disease seems to be clearcut, and only one feature is variable, namely, duration. Some cases may have a precipitous course (d'emblée type of Pernet), and others may be interrupted by remissions of several months. Two of their cases showed tuberculous adenitis. Pneumonia and nephritis were secondary. In one case in which inoculation of animals was done, a human strain of tubercle bacillus was recovered from a lymph gland which showed no histologic lesion characteristic of tuberculosis. (This raises the question: Does the presence of tubercle bacilli in tissue, without typical cellular reactivity on the part of the host, constitute true infection?)

A relatively large number of cases of disseminate lupus erythematosus having come under our observation in recent years, it was thought that a clinical analysis of these cases might be of value at this time.

REPORT OF CASES

Case 1.—Mrs. S. L., a white woman, aged 32, a housewife, was first admitted to Barnes Hospital in 1927 with the chief complaint of painful joints and an eruption on the face and hands of four months' duration. Previous to this illness she had enjoyed good health. She had been married for fourteen years and had one child in perfect health. There was a history of two induced terminations of pregnancy, following the second of which, in 1923, the patient was confined to bed for about six weeks with a probable pelvic cellulitis. Recovery was complete.

Four months before entering the hospital, she noticed a red "blotch" on the left side of her nose. One month later this began to spread over her cheeks and the lower part of her forehead. Erythematous areas also appeared on the hands. Three weeks before her admission to the hospital the hard palate became involved, the lesions finally spreading over the lips. Coincident with the extension of the process, the patient began to suffer from transient arthralgia, involving several joints in rapid succession without marked local signs of inflammation. There was a rapid loss of weight.

On her first admission to the hospital, the patient was obviously very ill. Her temperature was 104 F. with a corresponding pulse rate. The cutaneous lesions were typical of disseminate erythematous lupus. There was a characteristic single reddish plaque over the bridge of the nose, a "butterfly pattern" with the wings extending symmetrically down to within 1 cm. of the corners of the mouth. Outlying lesions were noted on the face, forehead, lips and chest. The palmar surfaces of both hands showed numerous plaques involving particularly the tips of the fingers and even extending under the nails. The lesions on the hands were reddish

purple and presented a shining surface with an eroded appearance in places. The lungs were clear on stethoscopic examination, the heart normal except for tachycardia, the blood pressure 112 systolic and 80 diastolic, and the liver barely palpable below the costal margin. Pelvic examination revealed a retrodisplacement of the uterus and a moderate degree of subinvolution. No disease of the adnexa was demonstrated. A laryngeal consultant found evidence of an acute maxillary and frontal sinusitis and an active pyorrhea associated with the lesions of the mouth. There were a few enlarged cervical lymph nodes.

The important laboratory observations on the first admission included: Stereoscopic roentgenograms of the chest showed a slight increase of the hilar

Fig. 1 (case 1).—Lesions on the face.

shadow on each side and a rather marked degree of hilar calcification. There was no gross parenchymal change, and the diagnosis was indeterminate. Roentgenograms of the sinuses were also reported as indeterminate. A series of roentgenograms of the gastro-intestinal tract showed colonic motor delay and evidence of a pathologic appendix. Repeated aerobic and anaerobic blood cultures were negative except on one occasion when a hemolytic type of Staphylococcus aureus was recovered. This was probably a contaminant. Several tuberculin tests in varying strengths were negative. The sputum was consistently free from tubercle bacilli. Daily urinalyses showed an occasional trace of albumin and a few white blood cells. There were no casts. The blood picture showed a definite leukopenia with a comparative increase in the transitional type of cells in the stained smear. The leukopenia was observed on repeated occasions, and was apparently uninfluenced by one transfusion of blood. A typical blood count was: red blood cells, 4,550,000; hemoglobin, 67 per cent, and white blood cells, 3,200,

with a differential formula of polymorphonuclear cells 69 per cent, lymphocytes 20 per cent, endotheliocytes 6 per cent and eosinophils 5 per cent.

The patient withstood a continual fever of the septic type, ranging as high as 105 F., with a pulse rate of 130, for three months. In seven weeks there was a loss of weight of 13 pounds (5.9 Kg.) in spite of a high caloric diet. Often at the height of her pyrexia she showed transient delirium and apathy. An ugly decubitus ulcer developed on the buttock. The patient suffered from a respiratory complication characterized by diffuse râles and marked dyspnea, and for a time she presented the clinical picture of bronchopneumonia. At the height of the fever the white blood cells were never above 8,000. There was a progressive anemia, the red blood cells reaching a low level of 2,600,000, with a hemoglobin content of 40 per cent. During the course of this devastating illness there was little change in the appearance of the cutaneous lesions. Under supportive treatment the patient improved, the temperature subsided, the symptoms in the chest cleared up

Fig. 2 (case 1).—Lesions on the hands.

almost completely, and the decubitus ulcer healed. After one week of normal temperature, at the end of three months of hospitalization, the patient insisted on being discharged.

Following her release, the patient's history was one of gradual decline. Pain recurred in the joints, and edema of the ankles was occasionally noted. She was unable to do any housework. At some time during the interim she suffered from an attack of an acute respiratory infection, diagnosed by her family physician as "pleurisy pneumonia."

In October, 1928, the patient consented to return to the hospital for one day for a check-up of her condition. The emaciation was more marked than it had been on her previous admission. Dyspnea was constant and was intensified to an almost unbearable degree on the slightest exertion. The respiratory excursion was greatly limited on both sides, and the respiratory rate more than 30 per minute. The respiratory minute volume was 10,400 cc. Against a pallid background, a suffused cyanosis was apparent. The breath sounds throughout both sides of the chest were high-pitched, without râles; there were dulness and diminu-

tion of breath sounds at the base of the right lung. On roentgen examination there was a fairly extensive, soft, parenchymatous mottling in the first and second interspaces on each side, probably more marked on the left. The right leaf of the diaphragm occupied a high position, and the costal angle was opaque. The patient undoubtedly had chronic mediastinitis. The heart was considerably enlarged or displaced, the apex being percussed about 12 cm. from the midsternal line in the fifth space. The blood pressure was 155 systolic and 110 diastolic as compared with the average readings of 110 systolic and 80 diastolic on the former admission. The liver and spleen were not palpable. There was no dependent edema. The extensive cutaneous lesions, which were a prominent feature of the previous examination, had faded and become scarred and atrophied in places to the fine texture of cigaret paper.

In the twenty-four hours during which the patient had agreed to remain in the hospital, the following laboratory data were secured: The roentgenogram of the chest was reported as suggestive of "pulmonary tuberculosis and pleurisy at the base of the right lung." Sputum could not be obtained for examination. The Wassermann and Kahn tests of the blood were negative. The nonprotein nitrogen content of the blood was 40 mg., and the blood sugar 132 mg. per hundred cubic centimeters. The electrocardiogram showed a tachycardia and left ventricular predominance. The vital capacity was only 500 cc., the average obtained in several trials in which the patient cooperated well. The basal metabolic rate was plus 7 per cent; the minute volume, 10.4 liters. Urinalysis showed: specific gravity, 1.020; albumin, 1 plus; hyaline and granular casts, and a few white blood cells and red blood cells. The guaiac test was positive. The blood count revealed a moderate anemia: red blood cells, 3,900,000; hemoglobin, 65 per cent, and white blood cells, 5,950, with the following hemograms: polymorphonuclears, 74 per cent; lymphocytes, 20 per cent; large mononuclears, 2 per cent; eosinophils, 2 per cent; transitionals, 1 per cent, and basophils, 1 per cent.

Final Admission to the Hospital.—On Dec. 21, 1929, the patient was readmitted to the hospital as an emergency case. She was dyspneic, orthopneic and almost moribund. The husband reported that the patient improved sufficiently after her last period of hospitalization to be up and around the house. However, a marked dyspnea was noted on exertion, and swelling of the ankles became marked during the final months before her readmission. She did not care for food. There were periods of nausea and vomiting. Her family physician gave her digitalis, without securing any improvement in the dependent edema.

Physical examination was difficult because of the extremely poor condition of the patient. Beneath the marked pallor one could see the pigmented scars of former erythematous lesions. On the previous admission to the hospital, extraordinary dyspnea and clinical evidence of mediastinitis were prominent symptoms. Now the marked edema of the left arm only, the displacement of the heart to the left and the presence of ascites suggested advancement of the mediastinitis with venous and lymphatic obstruction. Over the left pulmonary apex there were constant showers of râles; to a lesser degree, râles were also heard over the right apex. A diminution in breath sounds over the lower right side of the chest suggested a hydrothorax. No pericardial friction rub was heard, but pericarditis was suspected on the basis of the observations made on the previous admission; some signs were suggestive of the adhesive type of this disease. Cardiac decompensation, a gallop rhythm, a loud systolic murmur and an accentuated first sound were notable symptoms in addition to the cardiac enlargement and displacement. The blood pressure was 160 systolic and 100 diastolic. There was marked edema of the extremities.

Laboratory studies were—blood counts: red blood cells, 1,970,000; white blood cells, 12,500; hemoglobin, 30 per cent; Schilling differential count: segmented cells, 78 per cent; lymphocytes, 14 per cent; rod-shaped cells, 5 per cent, and monocytes, 3 per cent; urinalysis: cloudy urine, with a specific gravity of 1.020, acid and albumin 2 plus; microscopic examination: many white blood cells and several casts to a low power field.

In spite of heroic supportive measures, the patient died the day following her admission to the hospital. An autopsy was performed by Dr. H. A. McCordock.

Autopsy.—The anatomic diagnosis was: lupus erythematosus disseminatus, arteriolosclerosis, arteriolosclerotic kidneys (chronic diffuse nephritis of vascular origin), fibrous pericarditis, bronchopneumonia, calcified tuberculous foci in the lungs and tracheobronchial lymph nodes, fibrous adhesions between the visceral and the parietal pleura, edema of the gallbladder, periportal fatty infiltration of the liver and focal necrosis of the liver.

The body was that of a poorly nourished white woman, aged 32. Rigor mortis was not present. There was faint purplish-red bilateral pigmentation on the cheeks and continuous over the upper part of the nose. Numerous similar faint blotches could be made out over the chest and extremities. In a few places on the extremities there were pearly white scaly scars, but there were no fresh lesions on the skin. There was edema of the lower extremities and the left arm. The axillary and inguinal nodes could be felt but were not greatly enlarged. The mucous membranes were pale, and the teeth were in poor condition.

The abdominal cavity contained a few hundred cubic centimeters of clear yellow fluid. The liver showed a slight thickening of the capsule. In the pelvis the uterus was displaced backward. The other abdominal organs occupied their normal positions and seemed unchanged externally.

No free fluid was found in the thoracic cavity. Dense fibrous adhesions almost obliterated the pleural cavities. The visceral and parietal layers of the pericardium were adherent.

The pleura over both lungs was ragged where it had been pulled away from the wall of the chest. Small patches were free from adhesions, especially on the left side over the upper lobe. Both lower lobes felt boggy. On section the left lung showed an almost airless lower lobe. No definite areas of bronchopneumonia could be made out. The alveoli appeared to be filled with frothy edematous fluid, containing some blood, which gave the cut surface a solid red appearance. In places there were patches that were bright red and obviously represented areas of hemorrhage into the alveoli.

The upper lobe was edematous but did not show as much hemorrhage as the lower one. In the lower lobe of the right lung there was an old encapsulated caseous tuberculous focus. The rest of the lung tissue resembled that of the lower lobe of the left lung. In both the middle and upper lobes there was mostly air-containing tissue, but patches of collapse and areas of hemorrhage were seen.

The lymph nodes at the hilus of the lung contained old calcified caseous tuberculous lesions.

Both layers of the pericardium were adherent. The pericardium could be stripped from the heart only with difficulty because of the firm fibrous adhesions. The heart was enlarged, weighing 420 Gm. When opened, both ventricles were hypertrophied, especially the left, which measured 2 cm. in thickness. The edge of the mitral valve was thickened, but the leaflets were not distended nor were the chordae tendineae coarser than normal. All the other valves appeared normal.

Only small infrequent patches of yellow atheromatous thickening were seen in the aorta. Calcified lesions were not found.

The liver weighed 1,480 Gm. and measured 28 by 20 by 9 cm. Its capsule was thickened in places by a deposit of white fibrous tissue; elsewhere it was smooth. On section the lobular markings were distinct. Yellow zones could be seen at the portal spaces in some places. The centers of the lobules were deep red.

There was marked edema of the gallbladder. About 2 cm. of edematous tissue separated the bladder from the liver at its attachment. This edema could also be followed along the larger branches of the portal vessels. The gallbladder contained dark-colored bile. All the ducts were patent. No stones were seen.

The spleen weighed 180 Gm. The capsule was wrinkled and thickened but otherwise unchanged. On section the malpighian bodies were not distinct. The cut surface was a deep red, and the pulp was firm.

The pancreas was normal.

The gastro-intestinal tract showed no change. Its mucosa was smooth. No ulcers or scars were seen. The peritoneum was shiny. No changes were seen in the mesenteric nodes.

The kidneys were tiny atrophic scarred organs. The right one weighed 90 Gm. and measured 6 by 2.5 by 1.5 cm. Shreds of kidney substance came away when the capsule was stripped. The surface was finely granular with projecting yellowish-gray nodules and white depressed scars. On section the cortex was extremely narrow, measuring only about 2 by 3 mm. in most places. The normal striae of the cortex had entirely disappeared, giving place to blotches of gray and reddish-brown tissue. The large blood vessels had thickened walls.

The ureter, pelvis and bladder seemed normal.

The uterus, ovaries and tubes seemed of normal size, shape and structure and were not removed.

The brain was not removed.

Microscopic Observations: Sections of the kidney showed the most advanced changes associated with narrowing of the blood vessels. In the cortex there was no longer a normal arrangement of structures. Small groups of dilated convoluted tubules alternated with areas of scar. Most of the glomeruli were damaged to some extent. In the scars they were reduced to small hyaline bodies surrounded by atrophied tubules and fibrous tissue infiltrated with lymphocytes. Outside the scars hyalinized or partly hyalinized glomeruli were frequently seen. The afferent arteriole often displayed extreme hyaline thickening. In fact, the small arteries and arterioles usually showed this characteristic hyaline thickening.

The tubular epithelium was flattened, especially in the groups of dilated tubules that projected above the surface of the kidney. Pink-staining homogeneous casts filled many of the tubules. Some contained red blood corpuscles.

The larger blood vessels showed an ordinary arteriosclerotic thickening with reduplication of the elastic layer and fibrous intimal proliferation.

The muscular fibers of the heart were hypertrophic. There was an unusual fibrous pericarditis. The epicardium was greatly thickened by a layer of loose, edematous fibrous tissue. In the deepest layers of this, nearest the heart muscle, there were isolated patches of fibrin deposit with a few polymorphonuclear leukocytes. A section of the mitral valve was thickened by fibrous tissue at the free edge.

Many of the alveoli of the lungs were filled with fresh blood and edematous fluid, but there was no bronchopneumonia. The pleura was thickened by an increase in fibrous tissue.

A section through the liver and gallbladder showed to advantage the thick layer of edematous tissue about the gallbladder. This was a pink-staining coagulated homogeneous mass in which one could see here and there the nuclei of the connective tissue cells that had been pushed apart, the small blood vessels, nerves

and highly dilated lymphatics. The basal layer of epithelial cells in the gall-bladder stained well, but the superficial ones were necrotic.

In the liver there were areas of fatty infiltration about the portal spaces in many places. Small foci of necrosis were scattered throughout the section. They were seen in almost every part of the lobule. Collections of polymorphonuclears were seen about and within these necrotic foci.

There was no change in the pancreas aside from a hyaline thickening of the arterioles.

The epithelial layer of the skin was very thin, being made up of only two or three layers of cells. The papillary projections into the underlying connective tissue of the corium were very short. There was little tissue on the surface, and the whole section gave the appearance of a recent proliferation after an extensive destruction of skin, such as one sees following burns.

Summary of Autopsy: The lesions of lupus in this case were no longer present at the time of autopsy, but the skin examined showed a recent proliferation of the epithelial layer. By far the most serious lesions were in the kidneys. These were the type associated with arteriosclerosis, particularly a thickening of the tiny arteries or arterioles; in other words, the type of kidneys seen in cases of severe hypertension. Changes were also seen in the small vessels of other organs. There was a hypertrophied heart, but, of course, the pericarditis must also be held accountable for some of the hypertrophy. The immediate cause of death may have been heart failure, but it must have been sudden as there were no signs of chronic passive congestion in the organs.

Since the etiology of lupus erythematosus is still open to question, animals were inoculated with material from the most active looking areas of the skin, from the tuberculous foci in the lungs and from the kidneys. In all the animals receiving tissue from the lungs or lymph nodes, tuberculosis (human strain) developed. All the others, including those inoculated with skin, showed no change and were normal when killed six months after the injection.

Summary.—The disease had an insidious onset during a period of good health. The observations were: general malaise, arthralgia, septic fever with delirium and great prostration; a gradual appearance of widespread, typical, cutaneous lesions; persistent leukopenia and secondary anemia; remission, with a healing of the cutaneous lesions, atrophy and pigmentation; a tendency toward recurrent infections of the respiratory tract; nephritis; bronchopneumonic symptoms and death. Autopsy revealed fibrinous pericarditis, pleurisy and fatty infiltration and focal necrosis of the liver.

Case 2.—M. L. T., a white girl, aged 14, was first observed on March 5, 1927. At that time a number of erythematous papular lesions of an urticarial character were scattered over the face. She was given a soothing lotion and capsules containing extract of suprarenal gland. Two weeks later she was apparently well, as there were no lesions or any trace of lesions at the sites of the former eruption.

She was not seen again until May 19, or two months after she had apparently recovered. At this time she had a slight erythematous dermatitis over her cheeks. By June 1, this had developed into a "butterfly" redness over the cheeks and nose; This was the first suggestion of lupus erythematosus. By June 14, the eruption had become worse. The eyelids were edematous and the conjunctivae red; a definite diagnosis of lupus erythematosus was established. As it was suspected that the cause was gastro-intestinal toxemia, she was referred to Dr. D. L. Penny. On June 15, 22 and 29, she was given an intravenous injection of gold sodium thiosulphate. After the third dose she had a severe chill. On July 6, her face showed marked improvement, and on August 10, she was apparently well. The

skin of her face was perfect. No trace of the former eruption and no scar remained. She was next seen on Aug. 27, 1928, when Dr. Engman made the following note: "This girl comes in today with her hair dry and falling out in quantities and there is a thick silvery crust over the scalp. It does not seem to be lupus erythematosus but looks like it might be a dermatitis on a thyroid basis. The skin over the body is dry and scaly and she has some slight keratotic folliculitis."

The condition of the scalp was treated as seborrhea and cleared up by April 16, 1928, as did the cutaneous lesions.

On May 25, 1929, thirteen months after the previous visit, a keratosis developed on the helix of the left ear, which was probably caused by a tight hat. Radium was applied, and the lesion involuted within two months.

On July 13, she appeared with a marked acute attack of erythema multiforme. She had enormous symmetrical patches of typical erythema multiforme-like lesions on the trunk, arms, forearms, thighs and legs. The hands were swollen and the eyelids edematous. The lesions were erythematous wheals and annular erythema multiforme lesions. The eruption disappeared in about five days.

She was not seen again until August 9, when her face was involved in a macular, erythematous inflammation. There were a few crusted lesions in places, and she described bullae which she had ruptured. There were only a few of these, however. There was no scaling of the skin over these erythematous areas. The condition looked like a dermatitis which had been very acute but which was disappearing. The arms and forearms, however, showed a few erythematous macules, and there were also a few patches on the chest over the sternum but none on the rest of the body. She was again treated with the mild antiseborrheic remedy that had been used with success a year previously, and by August 16 the eruption was disappearing rapidly. Five days later, on August 21, she again appeared suffering with pain in the chest and fever, which she acknowledged she had been having for some time but had not reported. She was then referred to Dr. Howard Rusk for study.

The condition of her skin from March 5, 1927, to Aug. 21, 1929, may be summarized as follows: First, urticaria-like lesions appeared over the face, simulating acne, from which the patient recovered rapidly. Fifteen months later she returned with an acute "butterfly" dermatitis over the nose and cheeks, when lupus erythematosus was first suspected, and within two weeks was definitely diagnosed. She apparently recovered from the lupus erythematosus so far as the cutaneous manifestations were concerned, but on Aug. 27, 1928, reappeared with a scaling dermatitis over the scalp. She again recovered, but on July 13, 1929, was observed in a severe attack of erythema multiforme from which she recovered in five days. On Aug. 9, 1929, she had an acute erythematous inflammation of the face with the formation of a few bullae, and on August 21, the final crisis began.

On studying the various cutaneous manifestations from their inception, we were immediately struck by the fact that the patient had a generalized toxemia from some internal condition, and a diagnosis of acute fulminating lupus erythematosus was made.

The terminal illness began two weeks previous to her admission to St. Luke's Hospital, with slight chills and fever. She had had some nasopharyngitis, for which she was treated by a laryngologist. The fever, however, continued, and five days previous to admission a definite pain developed in the left lower part of the chest on forced inspiration. There was no headache, ocular symptoms or cough. The gastro-intestinal history was negative, as was the genito-urinary history.

It is interesting to note that this patient had a series of infections of the upper respiratory tract, the first being a "bronchial asthma" that occurred when she was 4 years of age, and persisted for two years; a tonsillectomy, in 1922, was followed by a stormy convalescence owing to a pulmonary complication, probably bronchopneumonia, and in 1928, shortly before the onset of the original cutaneous lesion, she had bronchopneumonia which lasted for six weeks. There was nothing else of note in the past history.

The family history relates that the paternal grandfather died of asthma, probably due to sensitization to cattle, and that the paternal grandmother had asthma at the time of the patient's illness. The mother and father were living and well. One sister was living and well.

Positive physical observations on the patient's admission to the hospital were: temperature, 101.2 F.; pulse rate, 100; respirations, 20; the right side of the nose was partially obstructed; there was generalized pharyngitis; the tongue was thick, coated and dry; there was limited expansion of the left side of the chest, and the percussion note was impaired; vocal fremitus and breath sounds were also diminished over this area. The edge of the liver was palpable at the costal margin. There was definite tenderness in the left upper quadrant of the abdomen, although the spleen could not be felt. The skin of the face was covered with a dull red macular rash with a fine branny desquamation in certain areas.

Report of the laboratory examinations follows. Blood count: white blood cells, 9,950; red blood cells, 3,730,000, and hemoglobin, 70 per cent; differential white blood count: segmented cells, 62 per cent; rod-shaped cells, 10 per cent; premyelocytes, 5 per cent; large mononuclears, 0.5 per cent, and lymphocytes, 27 per cent; nonprotein nitrogen content of the blood, 35 mg. Phenolsulphonphthalein test: 45 per cent in two hours. Urinalysis: albumin, 4 plus, but no sugar; a few white and a few red blood cells on microscopic examination. The Wassermann and Widal tests gave negative results.

The course at the hospital was progressive. The temperature was of the type seen in malaria, the range being from 99 to 106 F.; however, during the last forty-eight hours the temperature was sustained around 104 F. On admission there was a progressive diminution in the amount of urine, which developed into almost a total suppression during the last two or three days; during this time a generalized edema developed. The signs in the chest became typical of marked bronchitis, and signs of pleurisy at the base of the left lung increased; this was also confirmed by roentgen examination, which showed in addition some fine coalescent mottling in both apexes. All of these roentgen indications, in a more exaggerated form, were found in another film on August 27.

The white blood cell count steadily rose and fluctuated daily between 13,000 and 17,000, the polymorphonuclears increasing to 90 per cent. The nonprotein nitrogen content of the blood rose to 54.5 mg.; the creatinine amounted to 2 mg., and total chlorides to 377 mg. Three blood cultures were negative after incubation for ten days. Thick, yellowish sputum obtained during the last few days of illness revealed a large amount of pus and pneumococcus, type II. Daily specimens of urine contained a precipitate of albumin with a few white blood cells and an occasional coarse granular cast.

During the last seventy-two hours the pulse became irregular, and an electrocardiogram revealed short runs of auricular extrasystoles, the remaining complexes being normal.

On the tenth day after admission, because of the persistent tenderness in the left upper quadrant of the abdomen and the difficulty in palpation due to distention, a cystoscopic examination was made, which revealed a marked hemorrhagic cystitis,

especially about the trigone. Microscopic examination of urine from both ureters revealed many red and white blood cells and coarse granular casts. There were no bacteria.

During the entire illness the skin presented a generalized blotchy cyanosis. After the third day there were many vesicles, the size of a pinpoint, over the abdomen, neck and chest, filled with a clear fluid. Four days before death, there appeared on the skin over the coccyx and over the left calf anteriorly two dark blue areas, about 2½ inches (6.35 cm.) in diameter; these were definitely demarcated and flat, and the skin in the center was hard. The areas gradually turned black and were beginning to slough just before death. Unevenly distributed over the entire body there were also several bullae, about 1 cm. in diameter, containing a yellowish fluid which was negative to bacteriologic examination.

The treatment in this case was symptomatic and supportive. The patient was given three transfusions of blood to combat progressive toxic anemia, and drastic measures were used to stimulate diuresis. Treatment was of no avail, and on Sept. 5, 1929, fourteen days after admission to the hospital, the patient died of progressive cardiorespiratory failure. A postmortem examination was performed by Dr. L. S. N. Walsh.

Autopsy.—Examination revealed: acute cardiac dilatation; acute degenerative myocarditis; acute fibrinous and chronic fibrous pericarditis; ascites; edema of the legs and feet; edema and ecchymoses of the mucous membrane of the bladder, cecum and colon; congestion of the lungs, liver, spleen and kidney; bilateral purulent bronchitis; pleurisy and bronchopneumonia with abscess formation at the base of the left lung; a putrid sloughing ulcer over the coccyx; miliary granulomatous foci on the myocardium, liver, lung, pancreas, mucous membrane of the bladder, cecum and colon, and parovarian cyst. A culture of the heart blood was sterile. Cultures of the pericardial and pleural fluid were also sterile.

The granulomatous foci were described as follows: Frozen sections of the myocardium showed a few scattered small foci in which there were some deposits of fine fat droplets in the myocardial fibers. There were also larger droplets between the fibers. The pericardium was greatly increased in thickness by an active growth of young granulation tissue which was made up of young fibroblasts and numerous capillaries. There were hemorrhages scattered throughout this tissue. The surface was covered with fibrin in which there were relatively few polymorphonuclear leukocytes. Scattered throughout the myocardium were areas of infiltration, almost invariably around blood vessels in which polymorphonuclear leukocytes predominated. Large mononuclear leukocytes with vacuolated cytoplasm also occurred. Near the pericardium in many places there was a peculiar degeneration of muscle fibers which seemed to be replaced by a meshwork of fine fibers that stained deeply with hematoxylin, somewhat resembling mucoid tissue. Into such areas plasma cells had migrated in great numbers. The bulk of the myocardium, however, appeared normal. Cross striations were present in the fibers. Sections of pericardium stained with eosin and methylene blue (methylthionine chloride, U. S. P.) were negative for bacteria of any kind. The terminal bronchi were often filled with pus, and many of them were ruptured, with a discharge of the contents into adjacent alveoli; in this way, areas of bronchopneumonic consolidation were formed. There was a heavy fibrinopurulent exudate on the pleura, and the underlying lung tissue was solid. Many small abscesses occurred in the lung tissue just beneath the pleura. The vessels around the periphery of the lung near the involved pleura frequently showed an extensive perivascular fibrosis. There were scattered areas of atelectasis. Sections stained with eosin and methylene blue showed no bacteria.

Summary.—In a case of fulminating disseminate lupus erythematosus with marked constitutional symptoms, no evidence of tuberculosis, either pulmonary or glandular, was found. There were no lesions in the joints. Diffuse bronchopneumonia and nephritis occurred, followed by death. Autopsy revealed acute and chronic fibrinous pericarditis, pleurisy and granulomatous foci on the viscera.

CASE 3.—Mrs. A. W., a white woman, aged 48, entered Barnes Hospital on Aug. 23, 1929, complaining of pain in the joints, fever, malaise and an eruption which had started on the face about fourteen weeks before admission and which had gradually spread to the neck, upper part of the chest and extremities.

Her father was said to have died of tuberculosis at the age of 65; otherwise, the family history was irrelevant.

The patient had suffered from numerous infections of the upper respiratory tract in addition to the usual diseases of childhood. Noteworthy in the past history was the fact that she had had frequent attacks of urticaria, some of which were so severe that there was an associated syncope. The cause of this allergic state was never satisfactorily determined, and there was an apparent spontaneous recovery about fifteen years preceding her admission to the hospital. At the age of 46, the patient was operated on for an ovarian cyst. At the same time the left ovary and tube and the appendix were removed.

The winter preceding her admission to the hospital the patient noticed some generalized muscular pains, but these did not incapacitate her in her work as a waitress. During the following spring she first noticed small red spots around her finger-nails. The eruption next appeared on the lower anterior portion of the neck. After that the dissemination of the cutaneous lesions to involve the whole upper part of the chest, arms and face was rapid and was associated with symptoms of increasing malaise, anorexia, weakness and loss of weight. The condition finally became so severe that the patient went to a hospital. After a temporary improvement in the cutaneous manifestation, she began to lose ground again and was sent to Barnes Hospital.

The general physical examination revealed little outside of the eruption. There was evidence of loss of weight. The teeth were in poor condition. There was marked tenderness in the right upper quadrant of the abdomen, and the liver was palpable on deep inspiration. Examination of the heart and lungs showed no abnormalities, nor were any revealed in the pelvic and neurologic examinations. The blood pressure was 105 systolic and 76 diastolic. Examination of the skin revealed a macular erythematous eruption symmetrically distributed over the backs of both hands and fingers, and on the extensor and external surfaces of the arms and forearms. A similar area of cutaneous lesions was noted over the shoulders, neck and anterior part of the chest down to about the level of the fourth rib. The back of the neck and the face, with the exception of the upper lip, were entirely covered with the same erythematous eruption. The hair on the scalp was very thin. The eyelids were puffy.

The Wassermann and Kahn tests of the blood were negative. Pathologic indications in the urine included a trace of albumin and an occasional leukocyte. The erythrocyte count was 4,800,000, and the hemoglobin content 55 per cent. The leukocytes numbered 6,800, and the differential count showed polymorphonuclears 70 per cent; eosinophils, 1 per cent; lymphocytes, 19 per cent, and transitionals, 10 per cent. The Schilling count revealed: rod-shaped cells, 29 per cent; segmented cells, 48 per cent; eosinophils, 5 per cent; lymphocytes, 12 per cent, and mononuclears, 6 per cent.

Blood cultures showed no growth. Cultures of the skin revealed no pathogenic organisms.

Roentgen examinations of the chest showed slight evidence of pleurisy.

The patient's course in the hospital was progressively downhill. The temperature generally ranged from 100 to 103 F., with corresponding tachycardia. The patient appeared toxic, and complained of generalized muscular pain. She responded with difficulty to questioning, ate little, took forced feedings poorly and continued to have a septic temperature. Following Goeckerman's suggestion, roentgen therapy was given in the manner that he advised. Her condition improved somewhat. This was a false hope, however, for within two weeks she was completely prostrated again, and the lesions had spread rapidly over previously uninvolved areas. The patient suffered from extreme arthalgia; she kept her knees drawn up in bed; she could not extend her elbows beyond 90 degrees. She finally

Fig. 3 (case 3).—Eruption on the face, trunk, forearms and hands. The position of the arms was constantly maintained to relieve the arthritic pain.

died, apparently of toxemia, on Oct. 15, 1929. An autopsy was performed by Dr. McCordock.

Autopsy.—The anatomic diagnosis was: lupus erythematosus disseminatus, confluent lobular pneumonia, polyp of the colon, extreme fatty degeneration of the liver (periportal), focal necrosis of the liver (central), microscopic foci of carcinoma in the colon, absence of the left tube and ovary, old fibrous pelvic adhesions and localized acute peritonitis.

The body was that of an emaciated white woman, aged 48. There were numerous lesions over the skin of the face, neck, arms, hands and anterior portion of the chest. On the face the lesions were symmetrical and consisted of a thickening of the skin, which was purplish red. This thickened red skin occurred in

blotches, and in addition there were smaller macular eruptions. In many places there were also dried brownish crusts and silvery scars where lesions had apparently healed.

There was an old surgical scar in the lower abdominal wall. About 200 cc. of turbid yellow fluid was found in the abdominal cavity. The left tube and ovary and the appendix had been removed.

There were fibrous adhesions between the lungs and the wall of the chest especially over both lower lobes posteriorly. There was about 100 cc. of clear fluid in each pleural cavity.

The heart weighed 230 Gm., and measured as follows: mitral valve, 8.0 cm.; aortic valve, 6.0 cm.; tricuspid valve, 10.0 cm.; pulmonary valve, 6.5 cm.; left ventricle, 1.5 cm.; right ventricle, 0.7 cm. The epicardium was smooth. The valves were delicate, and there was no scarring of the myocardium or the endocardium. The aorta was smooth-walled, but contained a few yellow fatty patches.

The pleura was thickened over the lower lobe of the left lung. In places there was a thick layer of white fibrous tissue. Over the upper lobe there were a few tags of fibrous tissue attached to the surface near the apex. On section there was a widespread bronchopneumonic consolidation in the lower lobe, with scattered patches in the upper lobe. The pleural thickening on the right lung was not as marked as that on the left. There were ragged tags of fibrous tissue over the posterior surface of the lower lobe and in the apex. The middle and lower lobes were bound together by loose adhesions. On section the lower lobe was almost uniformly consolidated, and isolated solid patches were present in both the middle and upper lobes.

The liver weighed 1,700 Gm., and measured 26 by 19 by 10 cm. The capsule was smooth. On section the normal markings were accentuated by the presence of large yellowish areas uniformly and regularly distributed throughout the liver. From the gross appearance it could not be made out with certainty whether these were about the portal spaces or the central veins. These yellow zones were, however, clearly accumulations of fat in groups of liver cells. Between these fatty areas, normal-colored liver tissue was seen.

The gallbladder and bile ducts were normal.

The kidneys were about alike in size and appearance. The left one weighed 150 Gm. and measured 12 by 5 by 3 cm. On section the cortical striations were straight and regular. The pelvis was normal.

The pancreas weighed 110 Gm., and appeared normal.

The left suprarenal gland weighed 7 Gm. Both the left and the right glands appeared normal.

There were old fibrous adhesions between some loops of the small intestines, the sigmoid colon and the fundus of the uterus. A thin layer of fresh fibrinous exudate was present over the sigmoid and the loops of small intestine in the lower part of the abdominal cavity. Elsewhere the peritoneum was smooth. No source could be found for this localized peritonitis. The mucosa of the small intestine was normal. In the sigmoid colon, about a foot from the rectum, there was a small soft polyp that hung into the intestinal canal. The base of this was broad, and a cut through the polyp and underlying intestinal wall showed some thickening of the submucosa and an exudate over the peritoneum in this region.

The uterine mucosa was normal. The left tube and ovary could not be found, and there were dense adhesions on this side of the uterus. Both the ovary and the tube were found on the right side, but they were involved in the adhesions of the pelvis.

Microscopic Observations: One section of skin showed a thickened corium, which in places was infiltrated with a few lymphocytes and an occasional plasma cell. Where the epithelium remained, it was very thin and the papillary projections were shallow, extending only a short distance into the underlying connective tissue. This strip of epidermis had evidently regenerated recently and resembled that seen at the edges of healing burns or other lesions in which epithelium has been destroyed. In other places the epithelial cells were gone and the connective tissue of the corium was exposed. The exposed surface was composed of dense layers of necrotic connective tissue without any acute exudate. Polymorphonuclear leukocytes were scattered throughout the corium and were also encountered in small groups in the cut cutaneous fat tissue, especially near blood vessels.

There was a widespread, confluent, lobular pneumonia which was so extensive in one section that it could pass for a lobar involvement. Other sections showed a patchy consolidation. The exudate consisted of polymorphonuclears, red cells and fibrin, with an occasional clump of bacteria. Where the exudate was not plentiful, the alveoli contained a coagulated edematous fluid.

The heart was normal.

The liver showed extensive fatty degeneration. Only small groups of liver cells remained about the central veins. There was a great zone of cells about the periportal spaces, filled with fat. This was so extensive that at least two thirds of the liver cells seemed to have been destroyed. Foci of necrosis were numerous in the cells about the central veins, and in such areas polymorphonuclear leukocytes were found about the necrotic liver cords.

The kidneys and pancreas were normal.

The polyp from the large intestine was composed of tall columnar cells covering a delicate stalk of connective tissue that contained thin-walled blood vessels. Mitotic figures were found, but were not numerous. There was nothing in the polyp that suggested carcinoma. The base of the polyp with the attachment to the intestinal wall was not included in the section.

A section of colon showed a thick submucosa. The mucosa was normal. In the subperitoneal fat at one end of the section there was a small nodule of carcinoma. This was composed of groups of large epithelial cells. For the most part they were arranged in small nests of plugs; in places, however, there were well developed acini in which the cells were of a tall columnar type resembling those seen in the intestinal polyp. In places this carcinomatous tissue was found close to the peritoneal surface on which there was a thick layer of fresh fibrin; some strands of tumor cells were seen in the deepest layer of this exudate. Lymphatics of the intestinal wall contained small plugs of tumor cells.

Since no tumor, except the polyp, was seen at autopsy, it is difficult to determine the origin of the microscopic nodules of cancer in the intestine. One of two explanations seems possible: The polyp may have undergone malignant change at its base and infiltrated the intestinal wall; or at the operation, a year before, the tumor removed may have been a carcinoma and a portion of it left behind; the cancer found at autopsy would therefore represent a recurrence.

From its histologic appearance, the cancer resembled more an intestinal type than one arising in the ovary.

Postmortem sections of the skin were inoculated into guinea-pigs, with negative results after six months.

Summary.—Following frequent infections of the upper respiratory tract and attacks of urticaria, fulminating lupus erythematosus disseminatus supervened with

occasional remissions. Arthralgia occurred during the illness which ran a septic course, with pneumonia and nephritis, followed by death. Autopsy revealed acute peritonitis, fatty degeneration and focal necrosis of the liver.

Case 4.—Mrs. H. L., a white woman, aged 23, was first observed on Nov. 9, 1927, complaining of an eruption that had been present for two months. She had had no previous eruption of any kind. Examination revealed bright red, superficial erythematous plaques with superficial, thin, adherent scaling, distributed over the anterior portion of the face, especially under the lower eyelids. There were also a few lesions of a similar character on both elbows. A tentative diagnosis of early acute psoriasis was made, and as the patient was going to Chicago she was referred to Dr. Mitchell.

Fig. 4 (case 4).—Lesions on the face (courtesy of Dr. J. H. Mitchell).

On December 1, a letter was received from Dr. Mitchell from which we quote: "It appears to me that she has an exanthematous type of lupus erythematosus, and in my experience these have invariably ended fatally. She had one small dose of gold and sodium thiosulphate (25 mg.), with an apparent exaggeration of all of her findings. She is much worse than when she first came to me, but whether this is due to the treatment or in spite of the treatment, I am unable to say. She is returning to St. Louis." Another letter from Dr. Mitchell stated that he had given her two doses of sodium hyposulphite. "She was well on the way to a serious involvement before I gave her the gold and sodium thiosulphate, and after the injection she seemed to grow worse more rapidly."

We saw her again on December 4, when she stated that she had had a slight fever for the previous ten days, accompanied by swelling of the face. Examination

revealed that the entire face was involved in a bluish-red edematous process distributed with absolute symmetry. The eyelids were practically closed from edema. The mucocutaneous surface of the lips showed excoriations with thick crusts, and there were ulcerations in the mouth. Another feature of the case were the numerous vesicular, bullous and crusted areas at the sites of the former erythematous lesions. The lesions on the elbows were red but without scaling, and the same condition was present on the knees. She had had a spontaneous miscarriage about a week previous to her return to St. Louis. This apparently caused the eruption to flare up, with constitutional symptoms and fever ranging from 102 to 104.5 F. She was immediately sent to the Missouri Baptist Hospital.

The record there reveals that her family history was irrelevant and that she had enjoyed fairly robust health throughout her adult life. There was nothing in her past history which could be interpreted as having a bearing on the present illness.

The illness began three months before admission when a slightly reddened area appeared on the left upper eyelid. This gradually began to spread until it covered the oval of the entire face and extended nearly to both ears. It also appeared on both elbows, the neck and the back. It was red and somewhat scaly and itched. Three weeks before admission, the patient had a menstrual period which lasted for ten days and then ceased. One week later the flow started again, and the patient passed clots and pieces of tissue which she described as "membranes." For about ten days before admission, she noted edema of the face, and ulcers in the mouth and nasal cavity.

On physical examination the face was swollen, reddened and covered with small fine scales. Ulcers were present on the lips and on the hard palate. The pupils were reactive. The throat was normal. The heart and lungs were negative to routine physical examination. The blood pressure was 118 systolic and 80 diastolic. An abdominal examination gave negative results. There was a bloody vaginal discharge. Pelvic examination yielded negative results. The skin of the face was involved in an erythematous, slightly scaly eruption extending over the "butterfly" areas, but not much atrophy. There were small, circular, red areas on both elbows and the ulnar surfaces of the arm, which were covered by small scales.

Essential laboratory data were: blood count: white blood cells, 5,500, and red blood cells, 3,750,000; differential count: polymorphonuclears, 68 per cent; small lymphocytes, 5 per cent; large lymphocytes, 15 per cent; transitionals, 3 per cent, and monocytes, 9 per cent. A blood smear showed nothing abnormal. A Wassermann test of the blood gave negative results. A vaginal smear revealed numerous bacteria and leukocytes but no gonococci. The urine showed a slight trace of albumin and, on microscopic examination, numerous red blood cells.

The course in the hospital was one of steady decline. The temperature was continually of the septic type, from 101 to 106 F. The pulse rate ranged from 100 to 150. Respirations averaged 30 per minute. On the third day in the hospital it was noted that her condition was extremely critical. All of the lesions previously noted became reddened, excoriated and crusted, and the eyelids became very edematous. In spite of heroic supportive treatment, including a transfusion, the patient died on December 9. An autopsy was performed by Dr. H. N. Allen.

Autopsy.—The face was almost entirely covered with a fibrinous purulent exudate which came off as one continuous membrane, leaving a very red skin beneath. There were a few crust formations over the upper part of the chest and the anterior surface of the shoulders. The body was only fairly well nourished, but not emaciated. There were a few soft adhesions in the apex of each

pleura, and there was a slight degree of pneumonoconiosis. A few small completely healed tubercles were found in the lungs. The bases of the lungs were markedly congested. The heart was apparently normal. In the abdomen no adhesions were found. The abdominal viscera were congested, and there were a few hard tubercles in the spleen. There was a small blood clot in the uterine cavity. The uterus was small, and the cervix showed an abrasion and marked congestion.

Cultures of the heart's blood and uterus were negative. Sections of the skin showed desquamation of the epidermis and a subacute inflammatory process involving the corium.

Summary.—The course in this case of fulminating lupus erythematosus was septic; leukopenia, nephritis and pleurisy accompanied the condition. Death occurred two months after the first observation. Autopsy revealed healed tubercles in the lungs and spleen.

Case 5.—G. T., a white woman, aged 22, was first seen in the Washington University Dispensary suffering from infectious arthritis of unknown origin and general adenopathy involving especially the axillary nodes. There were no cutaneous lesions. She failed to improve under treatment in the clinic and was sent to Barnes Hospital on Jan. 15, 1921.

The family history was not significant. The past history revealed that the patient had had influenza in 1919, complicated by pneumonia, and that she had been confined to bed for nine weeks. Her present illness had begun three weeks before admission to the hospital, at which time she noted swelling of the right hand and wrist and a lump under the right arm. This arthritis later involved the shoulders and elbows. She had lost 15 pounds (6.8 Kg.) since the beginning of the illness.

Physical examination showed tenderness and swelling over the finger joints and right wrist, with limitation of motion of these joints and also of the right shoulder. The cervical and axillary nodes were palpable. The breath sounds were increased over the apex of the right lung, and the first heart sound was impure. The blood pressure was 95 systolic and 60 diastolic. Chronic tonsillitis and carious teeth were noted. A pelvic examination gave negative results.

Removal of foci by tonsillectomy and extraction of diseased teeth and appropriate therapy produced no result. A pleural friction rub developed, and a few râles were noted in the apexes of the lungs. These signs gradually subsided.

The patient had an irregular low grade fever during her stay in the hospital. After two months she was considered sufficiently improved to be discharged to a convalescent camp.

On this first admission important laboratory work included repeated urinalyses which yielded negative results. The tuberculin test was negative. The phenolsulphonphthalein test showed: first hour, 34 per cent; second hour, 14 per cent, and total amount, 48 per cent. Examination of the blood showed: red blood cells, 4,500,000; hemoglobin, 70 per cent; white blood cells, 5,200; polymorphonuclears, 64 per cent; lymphocytes, 15 per cent, and large monocytes and transitionals, 21 per cent. The test for sugar tolerance gave 91 mg. per hundred cubic centimeters for the first hour, 119 mg. for the second hour, 110 mg. for the third hour, and 111 mg. for the fourth hour. The complement-fixation test for tuberculosis was anticomplementary. Repeated cultures of the blood were negative. Pus from the teeth showed Staphylococcus albus and Streptococcus hemolyticus. The Wassermann reaction of the blood was negative. A roentgenogram of the chest was indeterminate. A 7 foot plate of the heart showed the organ to be of normal size.

On April 16, the patient was readmitted to Barnes Hospital with the following diagnosis: lupus erythematosus, simple anemia, stomatitis and acute nephritis. In

the convalescent camp the patient's symptoms in the joints had improved rapidly. On the evening of April 12, four days preceding admission, she noticed a slight reddening of the right forearm. The next morning her nose was "inflamed," and this inflammation spread to the cheeks. There were sensations of chills and fever without exacerbation of pains in the joints.

A physical examination did not show the patient to be acutely ill. There was a cutaneous lesion on the face, described as a "butterfly" area; this consisted of a number of slightly elevated flat papules with erythematous swelling over the nose and the adjoining areas of the cheeks. The entire area was moderately thickened. Cutaneous lesions of a similar character but more faded were noted over the right forearm and sternum. Over the back were several brownish discolorations with fine silvery scaling. The heart and lungs were essentially normal. The blood pressure was 132 systolic and 90 diastolic. Abdominal examination yielded negative results. The reflexes were active. In contrast with the previous admission, there was no evidence of involvement of the joints. Several days after the patient's admission to the hospital the lesions were described as irregular and reddish purple, with shiny and edematous skin. Similar conditions existed around the nares, especially the septum, and on the hard palate there was an irregular superficial ulceration with a grayish surface and a brilliant red edge. There was a sharply circumscribed area of pigmentation on the right forearm.

During the first two weeks in the hospital the patient ran an irregular temperature which ranged from normal to 101 F. During the last ten days this subsided almost completely. The lesions on the face and forearm were greatly improved. As the lesions disappeared, there was a deposit of brownish pigment.

On this admission the important laboratory observations included: Wassermann test, negative; nonprotein nitrogen, 38.7 mg.; phenolsulphonphthalein test, first hour 46 per cent, second hour 27 per cent and total amount 73 per cent. An average specimen of urine revealed hyaline and granular casts, and 3 Gm. of albumin per thousand cubic centimeters. The blood count showed: red blood cells, 3,530,000; hemoglobin, 70 per cent, and white blood cells, 6,900; the differential count revealed: polymorphonuclears, 48 per cent; lymphocytes, 37 per cent, and large monocytes and transitionals, 15 per cent.

Examination of the stool yielded negative results.

The patient was discharged to the outpatient department, but did not report there. She applied for admission to the Jewish Hospital on May 29. The following diagnosis was made: lupus erythematosus, ulcerative stomatitis, bronchopneumonia and acute nephritis. She gave a history of weakness, fever and sore mouth during the two and a half weeks following her discharge from Barnes Hospital.

When examined at the Jewish Hospital, she appeared acutely ill with a temperature of 102 F. and a pulse rate of 120, and there was an erythematous eruption over the face distributed in the "butterfly area" with poorly defined borders. The skin was thickened. The vermilion of the lips had a peculiar glazed appearance, and was evidently involved in the same process that affected the cheeks and nose. The mucous membrane of the entire palate, cheeks and tongue presented brilliant red patches studded with minute vesicles and tiny ulcers.

A general physical examination revealed palpable cervical lymph nodes. The heart and lungs were normal. The blood pressure was 120 systolic and 80 diastolic. The right lower pole of the kidney could be palpated. The inguinal nodes were moderately enlarged. The reflexes were normal.

The patient rapidly lost weight and declined in general. The lesions of the mouth grew progressively worse. On June 4, the liver was found to be palpable

about three fingerbreadths below the right costal margin. The spleen was not enlarged. Two days later the patient complained of severe abdominal cramps. There was no rectus spasm, and the white blood cells numbered 8,000. The patient soon became unable to take solid food because of the extensive lesions of the mouth (the smears from these showed Vincent's organism), but could take liquid nourishment. On June 8, diffuse râles were noted at the bases of the lungs, and although the edge of the liver was not felt, it was percussed as low as the umbilicus. The spleen was not palpable. Four days later the patient's fever rose from a previously high level of 102 F. to 104.4 F., and she complained of severe pain in the upper left portion of the abdomen and the left side of the chest, intensified by deep breathing. The signs in the lungs were those of a beginning bronchopneumonia; a systolic mumur developed over the precordium, and the pulse rate became rapid. The patient presented the clinical picture of a septicemia, although the blood cultures were negative. In spite of heroic supportive treatment, she died on June 29. Permission for autopsy was refused.

A summary of the laboratory observations at the Jewish Hospital is: On three occasions the Wassermann reaction of the blood was anticomplementary. Daily specimens of urine showed a large amount of albumin, a few white blood cells, a few red blood cells and occasional granular casts. The phenolsulphonphthalein test yielded 8 per cent for the first hour, 14 per cent for the second, and 22 per cent for the total amount. On admission, the blood count gave: red blood cells, 3,384,000; white blood cells, 5,200, and hemoglobin, 70 per cent. The differential count showed: polymorphonuclears 36 per cent; small monocytes, 54 per cent, and large monocytes, 10 per cent. The white cell count ranged from 4,400 to 16,000. The lowest value reached by the red cell count was 2,000,000, and that of the hemoglobin 55 per cent. The nonprotein nitrogen content was 35 mg. per hundred cubic centimeters. The Widal and paratyphoid agglutination tests gave negative results. Repeated blood cultures were negative. A smear from the throat showed Vincent's organisms. A culture from the throat yielded Staphylococcus hemolyticus.

Summary.—The patient had fulminating lupus erythematosus preceded by arthritis. The observations were: an irregular low to high grade fever, nephritis, bronchopneumonia, pleurisy, leukopenia and terminal leukocytosis, ending in death.

Case 6.—Mrs. N. U., a white woman, aged 35, a housewife, was admitted to the Barnard Free Skin and Cancer Hospital on Sept. 5, 1928. Her history stated that she had an eruption on the face, neck and arms which began eight months previous to her entrance to the hospital. One sister had died of tuberculosis, but the family history was otherwise negative. The patient had had measles and mumps in childhood, and an attack of influenza in 1918, with an uneventful recovery. There had been no systemic indications of importance except occasional coughs unaccompanied by night sweats, hemoptysis or extreme fatigue. There was nothing of interest in the menstrual history except that during the last two years there had been menorrhagia of two weeks' duration.

She had been married for twelve years and had had four miscarriages, each at the third month; one full term baby, a breech delivery, died at birth. There were no living children.

The onset of the present illness occurred about eight months before the patient's admission to the hospital. She stated that her ankles were swollen for about two months and then returned to normal size, before the cutaneous trouble began. The history was not suggestive of a constitutional cause for this edema. Next the face and eyes became swollen, the face flushing deeply. At about the same time there appeared a small dry rough spot beneath the left eye, which extended

over the entire face. This condition progressed, and in about a week the lesions were distributed over the neck and arms. Scaling and a burning sensation of the involved areas were prominent symptoms. Local applications failed to give relief. The patient had lost about 50 pounds (22.7 Kg.) during the year before her admission to the hospital.

On physical examination she appeared older than the stated age of 35, and she had obviously lost weight, but was not emaciated. Moderate seborrhea of the scalp was noted. The pupils were equal and reacted sluggisly to light, but were active in accommodation. The chest was thin, and the clavicular spaces were deepened.

Fig. 5 (case 6).—Type of eruption and distribution.

The percussion note was somewhat impaired over both apexes anteriorly, and the breath sounds were slightly diminished over the same area. No râles were heard. The heart was normal; the blood pressure was 102 systolic and 64 diastolic. Abdominal examination showed the liver about two fingerbreadths below the right costal margin, but otherwise gave negative results. The entire face, front and back of the neck, shoulders and extensor surfaces of the arms, forearms and hands were involved in the eruption. The skin of the face was dusky red, thickened, dry and scaly, with the exception of the upper and lower eyelids, the nasolabial furrows and the angles of the mouth, which were normal in color. There was considerable swelling over the front of the neck. On the extensor

surfaces of the arms and forearms were large plaques and discrete lesions, slightly infiltrated, pigmented and showing few areas of atrophy.

The essential laboratory data follow: A biopsy of a typical lesion from the arm showed: (1) a slight amount of hyperkeratosis; (2) edema, acute and chronic inflammation of the corium and normal vascularity; (3) thinning and an inflammatory reaction of the epithelium.

Blood counts during the early part of her stay in the hospital showed: red blood cells, 2,900,000; white blood cells, 10,000, and hemoglobin, 65 per cent, with the following differential formula: lymphocytes, 60 per cent; large mononuclears and transitional cells, 12 per cent, and polymorphonuclears, 28 per cent. During

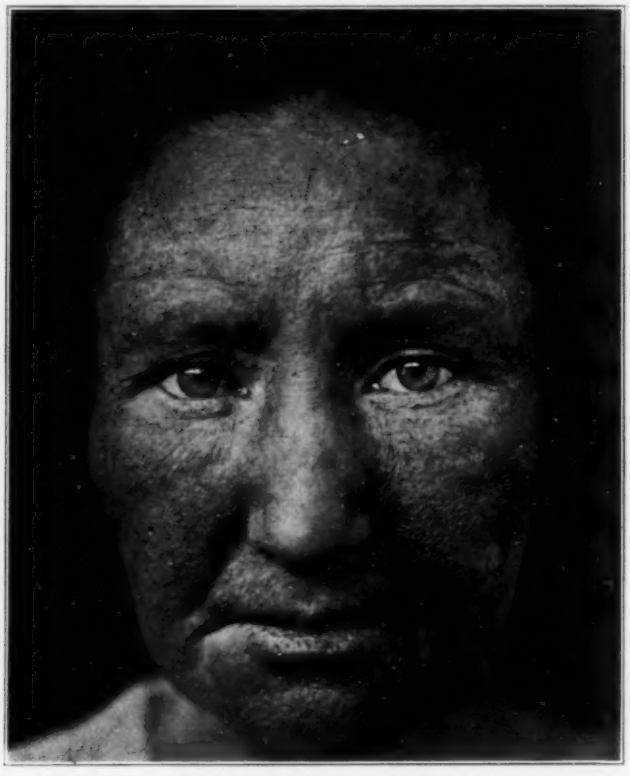

Fig. 6 (case 6).—Close-up of the eruption on the face.

the latter part of her stay in the hospital and during the course of the terminal pneumonia, the leukocytes rose to 28,000, and there was a predominance of the polymorphonuclear forms (85 per cent). Urinalysis yielded negative results on admission. As the patient failed, her urine showed an average albumin content of 3 plus and hyaline and granular casts. The Kahn test of the blood was negative. The Pirquet and intradermal tuberculin tests (with as much as 5 mg. of tuberculin) gave negative results. Repeated examinations of the sputum failed to reveal tubercle bacilli. Examination of the stool gave negative results.

The patient's temperature and pulse rate were normal during the first six weeks of her hospital stay, except for a slight reaction following the first two

intravenous injections of a gold preparation. The gold therapy seemed to cause an improvement in the lesions. On October 26, she complained of general malaise and, in particular, of pains in the back and in most of the joints. No elevation of temperature accompanied this complaint; the symptoms subsided, and three days later the patient was given her third injection of the gold preparation intravenously. A few hours following this medication there was a chill and a sharp rise of temperature to 104.5 F. The temperature remained at this high level, and two days later there were definite signs of consolidation in the bases of both lungs. There were bloody sputum, leukocytosis, dyspnea and great prostration. The

Fig. 7 (case 7).—Eruption on the face and neck, early stage.

appearance of cervical adenitis during the course of this diffuse pneumonia suggested to one consultant the possibility of an extensive mediastinitis. During the two weeks of high fever, there was a dramatic improvement in all the lesions, the redness, induration and scaling clearing up almost entirely, leaving areas of heavy pigmentation. The patient was digitalized and heroically treated for pneumonia, but after a period of temporary improvement she showed signs of a failing myocardium. Pulmonary edema ensued, and she died on November 24. An autopsy was refused.

Summary.—The patient had acute fulminating lupus erythematosus with a high septic fever, arthralgia, pneumonia, nephritis and clinical evidence of extensive mediastinitis, followed by death.

CASE 7.—S. O'K., a white woman, aged 28, entered Barnes Hospital on March 26, 1921. Her chief complaint was an eruption and swelling about both eyes, the face, chest and elbows, with generalized pains in the joints of three months' duration.

The family history showed no tuberculosis. The menstrual history was normal. The patient had been married nine years and had had six pregnancies, terminated by induced abortion. Her past history revealed the usual diseases of childhood, frequent sore throat and a tonsillectomy in 1919. There was no arthritis with the attacks of tonsillitis. She had uncomplicated influenza in 1918. Previous systemic indications were unimportant.

The patient dated the onset of her present illness to three months before, when an infection of the forehead, resulting from pulling out her eyebrows, was treated

Fig. 8 (case 7).—Eruption on the face and neck, six weeks later.

with a household disinfectant (CN, a crude carbolic derivative). This caused a spreading inflammation over the eyelids. Four weeks after the onset of this illness, there was pain in the wrist and elbow joints. At the same time, the inflammation spread over the nose and cheeks and finally involved the neck and upper part of the chest, the elbows and the right hand. Arthralgia involved several joints in rapid succession.

On admission the patient presented the following positive indications: temperature 100 F., pulse rate 82 and respirations 20. The lesions were described as follows: "The entire face, including both sides of the nose, the adjoining portions of the cheeks and both upper and lower eyelids, presented a peculiar dusky red inflammatory process showing dilated capillaries and, here and there, tenacious scales. The tip of the nose was free. The edges of the alae nasi were involved. The process also extended over both ears and down the neck. The lips were fissured and scaly. The posterior surface of both elbows showed similar lesions. On the dorsum of the right hand there was a thickened patch with some swelling

and papule formation. The nails on the fingers were thin." It was noted that the eruption associated with arthralgia and fever was characteristic of disseminate lupus erythematosus.

On general examination the thyroid was slightly enlarged, a few hard cervical lymph nodes were found, the clavicular spaces were deepened, the breath sounds were of a normal vesicular character, and the heart showed a soft systolic murmur over the apex. The blood pressure was 130 systolic and 90 diastolic. Abdominal examination yielded negative results. The shoulders, wrists and elbows were swollen but not reddened, and passive manipulation was limited by pain. Observations on the pelvic region were normal, as were the reflexes.

Fig. 9 (case 7).—Type of eruption on the hand.

The laboratory data were: The Wassermann test of the blood was negative. A series of blood cultures showed no growth. The nonprotein nitrogen content of the blood was 30.5 mg. per hundred cubic centimeters; the uric acid, 1.98 mg., and the creatinine, 1.37 mg.; blood calcium was 10.9 mg., and blood sugar 118 mg., per hundred cubic centimeters. An electrocardiogram was normal. The daily average specimen of urine showed a trace of albumin and occasional granular casts. The cardiac dimensions shown on x-ray plates were within normal limits. The white blood cell count ranged from 8,900, with an essentially normal differential count, to 20,000, with a large predominance of polymorphonuclear forms in the terminal stage of the disease, when pneumonia developed. On the patient's admission to the hospital, the red blood cell count was normal, and the hemoglobin content 88 per cent. During the course of the illness these values were reduced to red blood cells 2,300,000 and hemoglobin 45 per cent.

The patient's course in the hospital was one of steady decline. The arthralgia continued, and the lesions noted on admission also spread to involve the tongue and palate. The edema of the face and hands increased. Pain and stiffness in the jaws and neck made deglutition difficult. Finally swallowing of liquids became impossible, and acetone appeared on the breath and in the urine. Food was given through an indwelling nasal tube. The laryngologist found evidence of esophageal paralysis. On April 29, it was noted that the erythematous lesions on the face and neck were becoming pigmented a light brown color and were probably undergoing involution. Shortly afterward the upper half of the back and shoulders and the entire buttocks became involved in a bright red inflammation similar to that at the time of admission and probably of the same nature. Lesions on the neck showed evidence of superficial atrophy. Cultural studies on these lesions were negative. Later, atrophic areas appeared in the lesions over the body. A series of intravenous injections of typhoid bacilli seemed to cause a temporary improvement in the appearance of the widely distributed lesions. During her entire stay in the hospital, the patient had a low grade, irregular temperature, the fluctuations rarely rising above 101 F. During the last week, when a terminal pneumonia developed, the fever reached a peak of 104 F. Permission for autopsy was refused.

Summary.—A case of fulminating lupus erythematosus disseminatus, showing a rapidly developing anemia and arthralgia, ran a septic course, with esophageal paralysis, a profuse eruption, pneumonia and nephritis.

Case 8.—P. B., a white woman, aged 27, was admitted to the Jewish Hospital on May 24, 1923. She complained of an eruption which had been present for several years, but which showed no tendency to spread until the past year. There was a history of malaria and an attack of influenza complicated by pneumonia in 1922. For several years there had been frequent attacks of sore throat, accompanied by arthralgia. A nonproductive cough without night sweats, loss of weight or marked fatigue had been present during the past year. There was an indefinite history of palpitation and shortness of breath. No edema of the feet or other symptoms of cardiac incompetence were noted.

The present illness really began about nine years before. At the age of 18, the patient first noted an eruption on the face which occurred shortly after an attack of hives all over the body. About two and a half years after the onset, the eruption appeared on the arms. About one year before the patient's admission to the hospital, the eruption spread to the neck. There were no associated constitutional symptoms, but the patient believed that the lesions were more troublesome and prominent at the time of her menses. The patient was well nourished. Over the apex of the right lung posteriorly the breath sounds were harsh, but no râles were detected. The heart was within normal limits. Examination of the abdomen, pelvis and extremities yielded negative results.

Examination of the skin showed on the vertex an irregularly oval patch of alopecia (not total), about 5 by 3 cm. in diameter. The skin there was slightly thickened and had thick adherent scales. On the back of the scalp, near the hair line, was a large irregular laterally elongated area showing a similar condition, with islets of normal hair here and there. The skin of the face was generally thickened. There was a "butterfly" area over the lower half of the forehead and cheeks which presented an erythematous eruption, papular in patches, with occasional areas of slightly scarred atrophic skin. There were no nodules of the Leloir type, but the papules were unusually elevated to about 3 or 4 mm. On the chest the superior sternal and sternoclavicular areas showed an erythematous eruption with slight atrophy. There were no lesions on the upper arms. At the

elbows, particularly the left, and on the extensor surfaces only, there were rather sparse erythematous and elevated plaques. Some scarring and atrophy were present. On the back of the right hand and on the back of the right index finger at the knuckle there were several erythematous patches. The ends of the fingers were thickened.

Roentgen examination of the chest showed the apexes of the lungs clear; the hilar shadows were thickened and moderately fused with numerous small calcified areas. In the upper lobe of the left lung there were linear beaded shadows extending to the inner limits of the third zone. The conclusion reached was that a latent hilar tuberculosis and perhaps tuberculosis of the upper lobe of the left lung were present. Roentgenograms of the sinuses and teeth were negative. Fluoroscopic examination of the chest confirmed the foregoing observations.

The complement-fixation test for tuberculosis was negative. There was no reaction to 1 mg. of tuberculin injected subcutaneously. The Wassermann test of the blood was negative. Urinalysis showed no pathologic condition. The phenolsulphonphthalein test yielded 44 per cent for the first hour, 17 per cent for the second and 61 per cent for the total amount. Examination of the blood showed: white blood cells, 6,400; red blood cells, 3,360,000; hemoglobin, 75 per cent; polymorphonuclears, 71 per cent; small mononuclears, 13 per cent, and large mononuclears, 16 per cent. The coagulation time was six minutes. The nonprotein nitrogen content of the blood was 23 mg. per hundred cubic centimeters. A blood culture was negative. A culture of the tonsils revealed staphylococci. On microscopic examination, the tonsillar tissue showed chronic inflammation.

The patient was practically afebrile during her entire stay in the hospital. Under symptomatic treatment the lesions improved markedly. There was no complaint of pain in the joints. She was discharged from the hospital after recovery from a tonsillectomy, on June 6. Two weeks later, she reappeared at the office with a total relapse of the eruption, but subsequently she disappeared from observation.

Summary.—In a relatively mild case of lupus erythematosus a remission occurred. The patient had arthralgia and was afebrile. There was possible thoracic tuberculosis, with secondary anemia and slight leukopenia.

CASE 9.—Mrs. H. J. P., a white woman, aged 50, entered the Barnard Free Skin and Cancer Hospital on March 4, 1929, complaining of an eruption on the arms and legs.

The family history was irrelevant. The past history revealed the usual diseases of childhood, among them diphtheria at the age of 8. Scarlet fever closely followed the attack of diphtheria, and the patient had a stormy convalescence, spending an entire winter in bed. She had had frequent attacks of malaria, the last one when she was 15 years of age. She had had an attack of "influenza" several months before entering the hospital.

About six years before admission, she noticed red, rather flat areas of "eruption" on the face. These were not painful, did not itch and showed no tendency to spread to other parts of the body. She was advised to undergo a tonsillectomy to remove one obvious focus of infection, and this was done one year after the lesions had appeared. The eruption began to fade and finally disappeared. One year before admission, the lesions reappeared on the cheeks and nose, and the patient was confined to bed for a time with symptoms of general malaise diagnosed as "influenza." During this period of illness, slightly raised and reddened areas were noticed on the left wrist and on both arms and legs. She was then treated in the outpatient department of the Barnard Free Skin and Cancer Hospital where the diagnosis of disseminate lupus erythematosus was made. She received intra-

venous injections of typhoid vaccine and, during the following three months, a series of ten injections of gold and sodium thiosulphate. During this time the lesions improved greatly and her general condition became much better.

Physical examination revealed an obese white woman in no apparent pain or discomfort. The examination was negative except for evidence of extensive dental repair. Examination of the heart and lungs and of the abdomen revealed no abnormality. The pelvic floor was relaxed, and a moderate rectocele was present. The adnexa were apparently normal. On the cervix uteri was an erythematous plaque similar in appearance to a fresh lesion on the face. The blood pressure was 105 systolic and 80 diastolic. Numerous patchy irregularly outlined areas of erythema with desquamation were scattered over the arms, legs, abdomen and right side of the chest. Many healed lesions were present in the form of shiny thin atrophic patches.

The Wassermann and Kahn reactions of the blood were negative. The leukocyte count was 8,250 and the hemoglobin content 80 per cent. The erythrocyte count was 4,256,000. A differential count showed: polymorphonuclears, 60 per cent; small lymphocytes, 37 per cent; large lymphocytes, 1 per cent; transitionals, 1 per cent, and eosinophils, 1 per cent.

The patient did not react to 1 mg. of old tuberculin given subcutaneously, and the Schilling differential hemograms showed no significant change after this test.

Examination of the urine revealed no significant abnormalities.

Roentgen examination of the chest showed a slight increase of the hilar shadow and lung markings. There was no evidence of gross pulmonary change. The roentgen diagnosis was indeterminate.

The faded "butterfly" pattern on the face and the superficial thin atrophic scars of former lesions on other portions of the body were typical of healed disseminate erythematous lupus. The history of "influenza" associated with the erythematous eruption was characteristic of the severe arthralgia of the disease. The unusual feature of this case, which was apparently arrested when last observed, was that remissions had been induced during the preceding five years (1) by removal of an infected focus (tonsillectomy), (2) by nonspecific therapy (intravenous injections of typhoid vaccine) and (3) by intravenous injections of gold and sodium thiosulphate. There was no evidence of any active tuberculous involvement in this patient.

Summary.—In a relatively mild case, arthralgia was noted, fever was an insignificant symptom, and there was apparent recovery.

Case 10.—H. N. M., a white man, aged 60, was admitted to Barnes Hospital on Oct. 27, 1929, complaining of an eruption on his hands and general malaise and fever of several weeks' duration.

The family history was irrelevant. The past history revealed that in addition to the usual diseases of childhood he had had pneumonia at the age of 6, from which he made an uneventful recovery. He had had typhoid fever at the age of 29 which had left no residual damage. After that, with the exception of an occasional infection of the upper respiratory tract, he remained in excellent health until about six months before his admission to the hospital, when he was confined to bed for one week with an "influenzal attack."

His present illness began three weeks before admission with a spreading painful eruption on the left hand, followed in several days by a similar eruption on the right hand. Associated symptoms were headache, general muscular pains, weakness, anorexia and a loss of 10 pounds (4.5 Kg.) in weight. During the week preceding admission, pain in the neck resulted in a protective spasm of the cervical muscles, which was the patient's chief complaint when he entered the hospital.

Physical examination revealed a well developed and fairly well nourished white man. An eruption was present on the backs of the hands and fingers in the form of slightly red elevated papules and plaques without vesiculation. The teeth showed much dental repair. The tonsils were small and the pharynx normal.

The neck was held stiffly in one position, and there were resistance and pain on passive motion.

The thorax showed a moderate emphysema, but the lungs were entirely clear to stethoscopic examination. The heart was apparently normal, and the peripheral arteries were elastic for a man of the patient's age. The blood pressure was 135 systolic and 78 diastolic.

Except for a small, hard, rather nodular prostate gland, there was no physical abnormality.

The Wassermann and Kahn tests of the blood were negative. The nonprotein nitrogen content was 27 mg. per hundred cubic centimeters. The phenolsulphonphthalein test of renal function gave 55 per cent. Agglutination tests for Bacillus typhosus and Bacterium melitensis were negative, and a blood culture was negative. The blood count showed: erythrocytes, 5,020,000; leukocytes, 5,800, and hemoglobin, 75 per cent. The Schilling differential count showed: rod-shaped cells, 4 per cent; segmented cells, 72 per cent; lymphocytes, 20 per cent; eosinophils, 2 per cent, and monocytes, 2 per cent. The blood platelets were normal. The only pathologic indication in the urine was an occasional white blood cell.

On roentgen examination of the chest, the first portion and the arch of the aorta were prominent. The trachea was sharply deviated to the right. There were a slight increase of the hilar shadow and a slight general thickening of all pulmonary markings, indicating an increased air content. The diagnosis was aortic enlargement and emphysema.

During the first week in the hospital the patient had a temperature ranging to 101 F. in the afternoons, unexplained by the examination of the chest. There was no evidence of tuberculosis. The number of white blood cells on repeated counts remained low, usually about 3,000 to 3,500, regardless of the changes in temperature. Dr. M. F. Engman advised a high vitamin A, high caloric diet and cod liver oil. The only other medication was the elixir of iron, quinine and strychnine. The patient improved remarkably, the lesions cleared up, and the temperature returned to normal. He was discharged from the hospital on November 14 and disappeared from observation.

Dr. Engman diagnosed the case as disseminate erythematous lupus. His comment was: "While the eruption in this instance was confined to the hands, the case should be classified under the disseminate variety because of the classical findings of weakness, fever and apathy without other demonstrated cause and the rather characteristic skin eruption, plus leukopenia. Some other mild cases of lupus erythematosus disseminatus seem to be controlled by rest, high caloric and high vitamine diet, and we believe that this patient's recovery or rapid remission can be attributed largely to this therapeutic regimen."

Summary.—A relatively mild case of lupus erythematosus disseminatus showed anorexia, arthralgia, fever and leukopenia. There was apparent recovery.

CASE 11.—P. E. H., a white man, aged 29, was first observed on Aug. 6, 1928. He stated that the lesions first appeared on his hands in August, two years previously; later they had appeared on other parts of the body, the last place being on the nose. He had been carefully studied from a general standpoint at Johns Hopkins Hospital, but a definite diagnosis had not been made. The lesions that he presented were described as absolutely symmetrical, involving the entire backs of both hands; on each shoulder there were annular lesions up to 4 inches (14

cm.) in diameter. They apparently began as circinate lesions, coalescing to form gyrate patches, and the surface was slightly excoriated about the periphery as though there had been superficial bullous formation. There were a few lesions in the middle of the back and on the legs. The condition began as an erythema, with slight, soft, infiltrated patches or plaques which gradually enlarged. He had never been free from the eruption since it began. The eruption would almost disappear and then it would fully develop again within forty-eight hours. He appeared perfectly well and in fine physical condition. At this examination it was noted that the lesions greatly resembled lupus erythematosus, but they were not constant; they almost disappeared and then recurred. It was first thought that the condition belonged to the erythema group of skin diseases as described by Osler. On August 14, when he was seen again, lesions had appeared in the palms. On August 15, he was given 0.05 Gm. of gold sodium thiosulphate, and when again seen on August 23, he showed great improvement. It was then noted that the lesions looked more like lupus erythematosus. One of us (Dr. Mook) believed that it was lupus erythematosus of the Senear type with bullae. The patient appeared again at the office on September 10, quite ill. The lesions were much more inflamed, and he was sent to St. Luke's Hospital. On October 2, he again appeared at the office, and the following note was made: "He had been treated in the hospital for about two weeks with a high vitamine diet, and the effect could only be described as magical. He had had a high temperature and was quite sick. With this method of treatment, the temperature promptly fell, the eruption promptly disappeared and he made an apparent recovery. At the site of the former lesions deep pigmented macules with atrophy were left." Dr. Engman stated that this was undoubtedly a case of disseminate lupus erythematosus.

Roentgen examination of the chest showed a dense hilar shadow on each side, especially on the right, with a slight thickening of all lung markings, which had a tendency toward beading. In the first and second interspaces on the left side there was a moderate amount of fine, soft, parenchymatous mottling bearing a definite relationship to beaded lung markings. A similar change could be observed in the second and third interspaces on the right side. The bases of both lungs were clear. The cardiac shadow was within normal limits. The roentgen diagnosis was pulmonary tuberculosis.

Summary.—A relatively mild case with bullous lesions showed remissions. A septic fever occurred, and a roentgen diagnosis of pulmonary tuberculosis with no clinical signs was made. There was apparent recovery.

Case 12.—M. F. P., a white woman, aged 32, entered Barnes Hospital on April 19, 1926, complaining of an eruption on the face and body and general malaise.

The family history was of no importance and was negative for tuberculosis. The past history revealed that at the age of 13 she had had measles which the family physician said left her with "weak lungs." There was no history of persistent cough or loss of weight following this attack. During childhood the patient suffered from frequent attacks of sore throat. The menstrual history was normal. The patient was married at the age of 25; there was one instrumental full term delivery, the child dying in thirty-six hours. There was no history of an acute illness that confined the patient to bed during adult life, but she stated that during the fall and winter months she was usually troubled with "bronchitis," unaccompanied by fever and never productive of blood-tinged sputum. Chronic constipation was an associated symptom. The patient first applied to the outpatient department in 1916, for treatment for late syphilis, discovered during the course of routine examination. She was treated from March 18, 1916, to April, 1919.

The routine courses included mercury salicylate given intramuscularly and arsphenamine given intravenously. Following one injection of arsphenamine, the patient had a severe reaction which was not followed by an eruption.

In June, 1917, because of a history of hemoptysis, a roentgen examination of the chest was made, which showed thickening of the hilus and periphery, but this observation was reported as "indeterminate." In May, 1918, the Wassermann reaction was again reported three plus.

In December, 1925, the patient suffered from an earache and was given local applications, without relief. At about the same time she noticed that her cheeks were red and inflamed and there was a marked sensation of burning. The eruption which followed this sensation of burning was intermittent until one month before her admission to the hospital. The right ear-drum was incised by an outside physician and it continued to discharge pus until the time of her admission. For two weeks before she entered the hospital, the eruption had extended down to the waistline, involving the breasts and back. Earache, headache, insomnia and anorexia were associated complaints. Definite pain or disease of the joints was not noted.

The general appearance was that of an undernourished and acutely ill woman. The temperature was 100 F., the pulse rate 98 and respirations 20. A subacute process involved the entire face in front and laterally, extending on to the ears. The process was slightly scaly and somewhat thickened. There was a sharp demarcation between the inflammation and the apparently healthy skin surrounding the eyelids. This peculiar absence of an inflammatory process was also seen around the lips, where there was a sharp line of demarcation between the healthy and inflammatory skin. The same condition existed in the skin about the nares. On the forehead the inflammatory process, instead of being sheetlike, was reticulated by small areas of apparently normal skin. The inflammation of the nose was accompanied by some atrophy. On the sternum and in the right clavicular area were several circular scaly patches from 0.5 to 1 cm. in diameter, the centers of which were atrophic. Between the scapulae were similar patches showing atrophy, and farther down on the back were a few smaller areas. A dry seborrhea of the scalp was present. A small chain of cervical lymph nodes was palpable. In front of the right ear there was an inflamed red area, and there was tenderness over the mastoid.

An otologist described the condition of the ear as a perforation of the drum with a discharge of thick pus. There was marked otitis externa.

Examination of the chest showed slightly impaired resonance at the apex of the left lung; the breath sounds were harsh with an increased whisper and a few inconstant râles at both apexes. The heart was apparently normal. Abdominal examination gave negative results except that the lower edge of the liver was palpable. The pelvic region was normal.

Important laboratory observations follow. The Wassermann reaction of the blood was negative. The nonprotein nitrogen content was 31 mg. per hundred cubic centimeters, the blood sugar 103 mg. and the uric acid 5.7 mg. The excretion of phenolsulphonphthalein was 10 per cent after the first hour and 35 per cent after the second. The tuberculin test was inconclusive. The subcutaneous injection of tuberculin gave a doubtful reaction. On repeated examination, no tubercle bacilli could be demonstrated in the sputum. Many pus cells and bacteria of all kinds were noted.

Roentgen examination of the chest showed an increase of the hilar shadow, thickened lung markings and soft parenchymatous mottling, best observed in the first three interspaces on the left. The diagnosis was pulmonary tuberculosis.

Other pictures showed "right frontal and ethmoid sinusitis, bilateral maxillary sinusitis and bilateral mastoiditis."

Examination of the blood showed: red blood cells, 3,990,000; white blood cells, 9,500; hemoglobin, 60 per cent; polymorphonuclears, 67 per cent; lymphocytes, 26 per cent, and transitionals, 7 per cent.

Examination of the urine showed albumin 1 plus, with occasional granular casts and few white blood cells and red blood cells.

The course in Barnes Hospital was uneventful. The rectal temperature ranged from normal to 100.5 F. Treatment was symptomatic, consisting chiefly of local

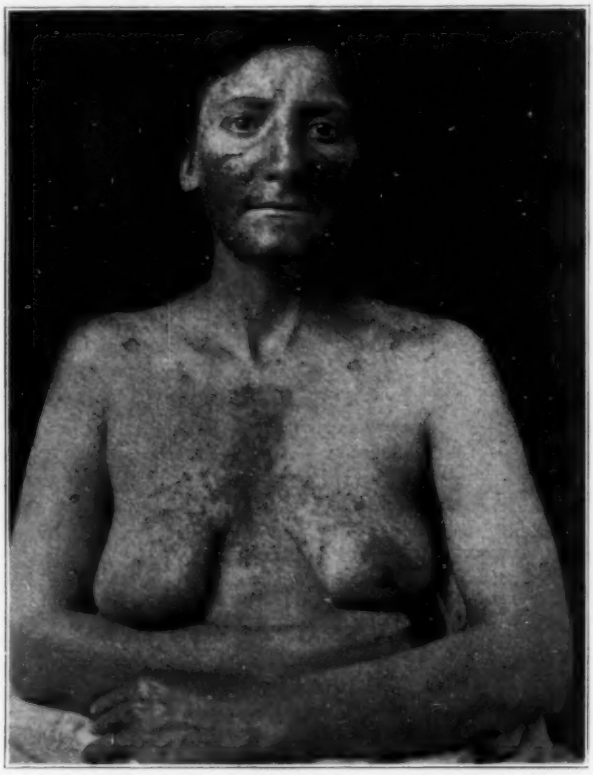

Fig. 10 (case 12).—Type and distribution of eruption, early stage.

applications to the lesions and analgesics. The patient was recommended for sanatorium care and was discharged from the hospital on May 5, 1926.

About six weeks following her discharge from Barnes Hospital, the patient was admitted as an emergency case to the Barnard Free Skin and Cancer Hospital, on June 21, 1926.

During the interval she had shown increasing weakness and general malaise. She thought that the lesions had grown worse since her discharge from Barnes Hospital. General physical examination revealed about the same condition as that described previously. In addition, the patient had lost considerable weight, and the lesions were of a brighter red and were widely disseminated over the forehead, nose, cheeks, chin, ears and temporal region. On the anterior and posterior sur-

faces of the chest, abdomen and back there were slightly raised and red scaly papules with considerable scarring in the central portions.

Urinalysis showed: albumin 3 plus, pus cells and granular and hyaline casts. The blood count was: red blood cells, 3,600,000; white blood cells, 14,500; hemoglobin, 68 per cent; polymorphonuclears, 92 per cent; small lymphocytes, 7 per cent, and large mononuclears, 1 per cent.

The patient's course in the hospital was one of rapid failure. On the ninth day her temperature rose to 104 F., her pulse rate to 120 and respirations to 34. The temperature remained elevated and septic for the last eight days of her life.

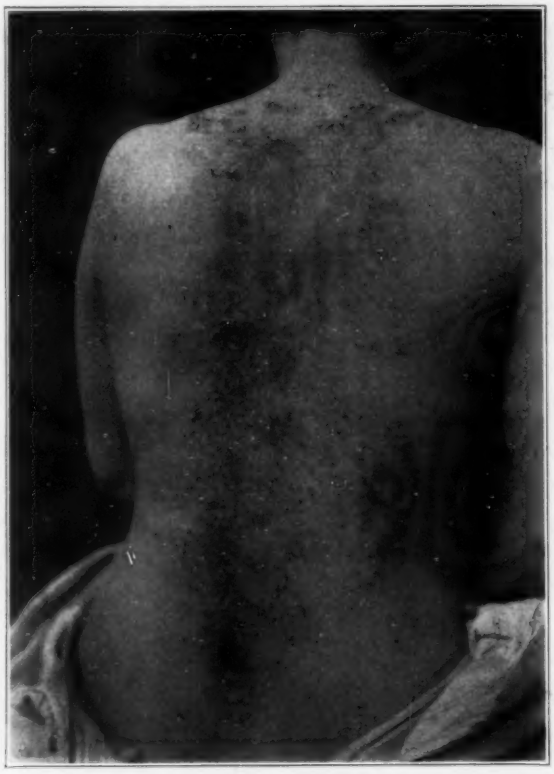

Fig. 11 (case 12).—Type and distribution of the eruption, early stage.

On the day preceding death, the following comment was made: "This case has been changing in the last week from that of disseminated discoid patches to a distinct tendency to bullous eruptions like those of septic pemphigus. Bullae have appeared here and there, of a flaccid character, not symmetrical, surrounded by an areola. On July 5, the skin seemed definitely worse and was already showing changes seen in septic pemphigus, with some hemorrhages from the gums and the nose. The patient is in an apathetic condition, and there are reddish purple bullae over parts of the entire body. Half of the left breast and the surrounding chest are denuded of epidermis. These bullae appeared before any intravenous injection of mercurochrome, which caused only a temporary fall in temperature and severe dysentery. The case looks very much like those fatal ones of vaccine pemphigus, especially the mouth with its bloody discharge."

The patient died on July 8. Permission for an autopsy could not be obtained.

Summary.—A fulminating case of the Senear-Usher type was preceded by numerous infections. Erythematous lesions changed to the bullous type. Slight adenopathy was present. A short remission took place. The fever and course were septic. There was slight clinical and roentgen evidence of tuberculosis, and nephritis was observed. The patient died.

Case 13.—Mrs. T. A., a white woman, aged 50, was admitted to the Barnard Free Skin and Cancer Hospital on Jan. 6, 1922, complaining of an eruption over the face and body.

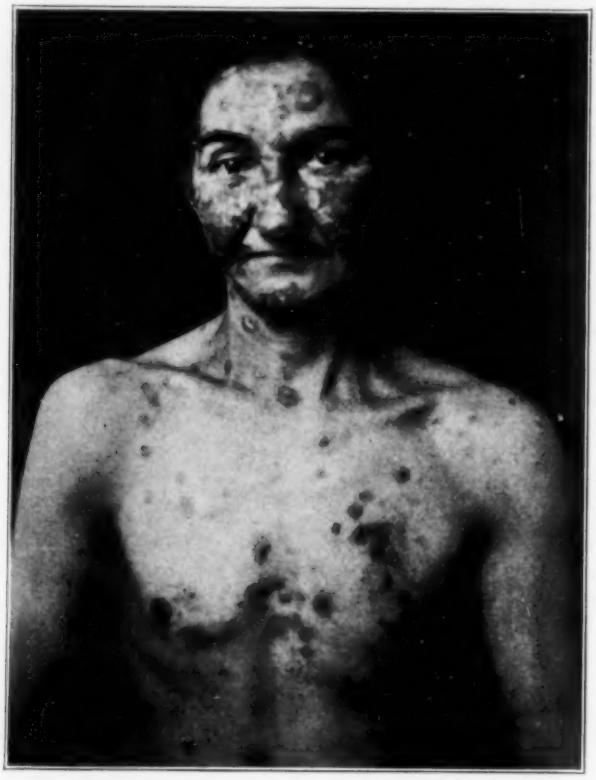

Fig. 12 (case 13).—Type and distribution of the eruption, early stage.

The family history was irrelevant. The past history revealed that she had had measles when 12 years of age. There were no other complaints except moderate constipation.

The present illness began two years before admission when the menopause started. At that time the patient noted a small brown elevated slightly scaly plaque on the right lower portion of the abdomen. This was treated with local applications, and it disappeared. A month later a small lesion, which was described as a "pimple," appeared on the right cheek; the condition soon spread over the nose and face, and small circular areas appeared on the body.

Examination revealed an inflamed, indurated, scaly, plaquelike lesion over the nose and cheeks in the "butterfly" areas. There were numerous circinate, margi-

nate and gyrate confluent patches over the forehead, chest and chin. The lesions on the face and forehead were covered with yellow oily scales which were slightly adherent. There was no bleeding when they were removed. Profuse lesions on the body were covered with scales which were piled up thickly, somewhat resembling syphilitic lesions except for their color, which was yellowish.

Physical examination showed the patient to be fairly well nourished in spite of the extensive lesions. Examination of the chest by Dr. J. J. Singer revealed no evidence of pulmonary tuberculosis on either physical or roentgen examination. The hilar glands were somewhat enlarged and the markings thickened in the upper lobe of the left lung, but in Dr. Singer's opinion that was insufficient evidence on which to base a diagnosis of tuberculosis.

The Wassermann test was repeatedly negative. Biopsy revealed lupus erythematosus.

The patient passed from observation during May and June, 1922. In May, a bullous eruption developed, the formation of new bullae being preceded by chills. On July 21, she was readmitted to the hospital with a generalized erythematosquamous and bullous eruption. Remains of ruptured bullae, several of which were covered with yellow crusts, were scattered over the chest, back and face. There was a great amount of desquamation. A discharge from both ears developed.

Examination of the urine showed a large amount of pus and few casts. During the last admission to the hospital the patient had a very low grade fever (from 99 to 101 F.), and the pulse rate averaged 100. Examination of the blood showed: red blood cells, 5,760,000; hemoglobin, 80 per cent; lymphocytes, 20 per cent; polymorphonuclears, 63 per cent; mononuclears, 11 per cent, and eosinophils, 5 per cent.

She left the hospital against the advice of the staff on August. 10.

On April 16, 1925, the patient was admitted to the St. Louis City Hospital, where she died. Permission for autopsy was not obtained.

This case was reported (with photographs) by Senear and Usher.[7]

Summary.—In a chronic case of the Senear-Usher type the lesions changed from the erythematous to the bullous type, and finally dermatitis exfoliativa appeared. The patient lived for six years, showing a septic course at intervals. There were no remissions.

ANALYSIS OF CASE REPORTS

All of the thirteen patients with disseminate lupus erythematosus herein described were of the white race; eleven were females and two were males; this predominance of the female sex is in accordance with previous reports. The ages varied from 14 to 60, the distribution by decades being one in the second decade, five in the third, three in the fourth, three in the fifth and one in the sixth. The great majority—eleven patients—were between the ages of 21 and 50, and eight were between the ages of 21 and 40.

Duration.—Of the thirteen patients, nine died and four disappeared from observation. In six of the nine fatal cases the total duration of the disease was one year or less (three months, five months, six

7. Senear, F. E., and Usher, B.: An Unusual Type of Pemphigus, Combining Features of Lupus Erythematosus, Arch. Dermat. & Syph. **13**:761 (June) 1926.

months [two cases], eleven months and twelve months). The other three lived for two and a half, three and six years, respectively. In other words, the disease was fatal within a year in two thirds of the fatal cases. The four patients whose fates are unknown had had the disease for from several weeks to nine years.

Family History.—The family histories furnished no information except in two cases, in one of which it was stated that the father had died of tuberculosis and in the other that a sister had died of tuberculosis.

Past History.—The past history showed that many of the patients had had infections of the upper respiratory tract (many of them on numerous occasions) and focal infections. The history of influenza was given rather frequently, and in some of the cases may not have been true influenza; instead the symptoms may have been caused by the beginning erythematous lupus.

Eruptions.—The eruptions noted in this group of cases may all be classified under the erythema group of Engman and Weiss.[8] Urticaria, wheal-like papules, erythematous plaques, erythema multiforme, erythema bullosum, pemphigoid eruptions and even a tendency to purpura were noted. The polymorphism of the cutaneous manifestations has previously been well described, notably by Goeckerman, Keefer and Felty, MacLeod and Sequeira.

Remissions.—Remissions of the disease occurred in all of the cases except the following three: case 4, a fulminant case, in which the patient died three months after the onset; case 6, in which the patient died in eleven months, and case 7, in which the patient died in five months.

Symptoms of the Joints.—Symptoms of the joints were one of the most prominent features of the series. There were severe arthritis in one case, varying degrees of arthralgia in eight cases and no symptoms in the joints in four cases. In two of the latter cases the presence or absence of symptoms in the joints was not noted. We consider these symptoms to be of great importance and regard them as manifestations similar to those considered later under the heading of involvement of the serous membranes. It is unfortunate that none of the joints was opened at autopsy in any of our cases.

Fever.—In two of our cases, fever was absent or insignificant (cases 8 and 9). In all of the others a septic type of fever had been present and in some to an extreme degree. The extremes of temperature were noted, as a rule, in the terminal stages when secondary infection, bronchopneumonia, etc., had occurred.

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8. Engman, M. F., and Weiss, R. S.: Erythema Group of Skin Diseases, Arch. Dermat. & Syph. **12**:325 (Sept.) 1925.

Adenopathy.—Adenopathy, evidenced by clinical examination, was not a feature of this series. General adenopathy was noted in case 5, and marked cervical adenopthy in case 6; cervical adenopathy was mentioned in cases 7 and 12. In the remaining cases there was no adenopathy found or none noted on the records.

Tuberculosis.—Definite clinical evidence of tuberculosis was rarely found. In case 8, a diagnosis of possible latent hilar tuberculosis and suspected tuberculosis of the upper lobe of the left lung were noted as the result of physical and roentgen examination. In case 9, a diagnosis of pulmonary tuberculosis was made from the roentgenogram, and in case 12 there was a questionable diagnosis of tuberculosis. Thus, in three of thirteen cases, clinical and roentgen evidence warranting a suspicion of tuberculosis was found. No tubercle bacilli were found in the cases in which sputum was obtained. Clinically demonstrable glandular tuberculosis could not be demonstrated in any of the cases. Four cases in this series came to autopsy. In cases 2 and 3 no tuberculosis was found. In case 1, calcified tuberculous foci were found in the lungs and tracheobronchial lymph nodes; in case 4, there were healed tubercles of the lungs and spleen.

In case 1, a human strain of tubercle bacillus was obtained from the lung tissue and mediastinal lymph nodes by inoculation of guinea-pigs. In case 3, postmortem sections of skin were inoculated into guinea-pigs with negative results after six months' observation.

The few tuberculin tests performed in this series were of no significance.

Nephritis.—In the nine patients who died, renal damage varying from mild nephrosis to severe nephritis was found. This was of the toxic type and evidently secondary to the profound toxemia or complications of the disease. The four cases that disappeared from observation presented no evidence of damage to the kidneys.

Examination of the Blood.—Leukopenia, frequently noted by other observers, was noted in five of the thirteen cases and secondary anemia in nine. Leukocytosis usually followed the leukopenia when complications and terminal infections developed.

Involvement of Serous Membranes.—The importance of this consideration has already been suggested, and is perhaps the most striking feature of the series. In ten of the thirteen cases, involvement of the serous membranes was either proved or convincingly suggested. In case 11, in which there was a seeming recovery, no evidence of serous involvement was found. It is striking that in all three of the cases of the Senear-Usher type (cases 11, 12 and 13) no evidence of serous involvement was found. This fact, it seems to us, is the only serious consideration against classing the latter type of case as disseminate

lupus erythematosus. We have gained the impression that Senear and Usher regard their cases as atypical pemphigus, while we are inclined to class them as lupus erythematosus disseminatus with pemphigoid features. Bullous lesions have been noted by many observers (Hallopeau [9]).

Arthralgia was definitely present in eight cases and possibly present in one other.

Pleurisy was definitely found in five cases and was suggested by the mediastinitis in case 6.

Peritonitis was present in cases 2 and 3.

The important observation of pericarditis was made in cases 1 and 2 and was suggested by the mediastinitis in case 6. Pericarditis was observed by Keefer and Felty in one of their cases, but the observation was not stressed.

Damage to the Liver.—Fatty infiltration and focal necrosis of the liver were found in cases 1 and 2.

Pneumonia.—This complication was present in six cases (cases 1, 2, 3, 5, 6 and 7). It is frequently the terminal feature.

COMMENT

Our study of this series of thirteen cases reveals clinical observations that are practically in agreement with previously published reports in many respects.

Clinical evidence of tuberculosis was found in only four of the cases. This evidence was strikingly meager in all four cases, and active tuberculosis could be excluded. Clinically demonstrable glandular tuberculosis was absent from the series. Autopsy disclosed calcified tuberculous foci in the lungs and tracheobronchial glands in one case, and similar lesions in the lungs and spleen in another case. No tuberculosis was found in the other two cases that came to autopsy. We feel that tuberculosis, if it plays a part in the etiology of the disease, must do so in a manner hitherto not described. Perhaps as Goeckerman suggested, "tuberculosis creates an allergic background and septic infection periodically excites explosions in the predisposed and unstable organism." If tuberculous infection creates such an allergic state, the majority of city dwellers are in danger of acquiring disseminate lupus every time they acquire a septic infection. Opie [10] suggested that practically all dwellers in large communities have or have had tuberculosis. With such an incidence of infection, we would expect to see a greater number of cases of disseminate erythematous lupus.

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9. Hallopeau: Ann. de dermat. et syph. **2**:389, 1891.

10. Opie: Papers read before the Washington University Medical Society and the St. Louis Medical Society. One paper was abstracted in J. Missouri M. A. **14**:87, 1917.

We succeeded in cultivating a human strain of tubercle bacillus from the pulmonary tissue and mediastinal lymph nodes in case 1. The tissues were calcified foci of latent tuberculosis. This had previously been done by others, and we do not attach any etiologic significance to the fact. We believe that it could be done in a very large percentage of general necropsy material.

Adenopathy has been stressed as an important symptom. It was apparently of minor importance in our series.

We regard leukopenia as an important symptom. It has been noted by others, but we do not believe that it has been sufficiently stressed as a diagnostic point.

The involvement of the serous membranes is an outstanding observation of this series. Arthralgia (serous and fibrinous arthritis?), pleurisy, pericarditis and localized peritonitis were found in ten cases of the series; in two cases in which serous involvement was not found, the symptoms were relatively mild, one of these patients apparently recovering.

We are inclined to agree with MacLeod, Keefer and Felty that disseminate erythematous lupus is a toxic or a septic cutaneous symptom.

The frequency of tuberculous infection and the rarity of disseminate lupus erythematosus should preclude the probability of the tubercle bacillus being the etiologic factor.

SUMMARY

Thirteen cases of lupus erythematosus disseminatus are reported, three of which are of the Senear-Usher type.

Nine of the patients died, and four passed from observation so that their ultimate fate is unknown.

Four of the cases came to autopsy, and the results of the autopsies are reported.

In six of the nine fatal cases, the duration of the disease was less than one year. The other three patients lived from two and one-half to six years.

Remissions were a feature of most of the cases.

Fever, septic in character, was a marked symptom in eleven of the cases.

Leukopenia is stressed as an important diagnostic symptom.

Adenopathy was not a prominent symptom.

The etiologic relationship of the disease to tuberculosis is discussed, and the opinion is advanced that there is insufficient evidence to substantiate this theory of the etiology.

Attention is called to the frequent involvement of the serous cavities of the body.

ABSTRACT OF DISCUSSION

DR. W. H. GOECKERMAN, Rochester, Minn.: My own experience harmonizes with the observations made by the authors. The various manifestations speak for a septic process, but do not exclude the bacillus of tuberculosis as a factor in some or possibly all cases. The rarity of disseminate lupus erythematosus and the frequency of tuberculosis do not exclude each other. True tuberculids are rare also, and tuberculosis is common. The biologic considerations in tuberculosis, especially those related to immunity, are as complex as those of syphilis; yet the etiology of lupus erythematosus is entirely unknown, but the tuberculous factor is not excluded. I am glad that the authors again called attention to the frequency of leukopenia; this change in the blood picture is also common in our material; at times it is marked.

DR. SAMUEL AYRES, JR., Los Angeles: I think that Dr. Mook is to be highly congratulated on the detail with which he has presented this subject. I think that we have concentrated so highly on the theory of tuberculosis that we have been blind to all other possibilities. On checking over his cases, I cannot help feeling that possibly the matter of focal infection has been of greater moment than he is willing to state. Eight of the thirteen patients gave definite evidences of upper respiratory tract infections. In this connection it is interesting to recall the paper of Dr. Madden at the Philadelphia meeting, in which he reported several cases of lupus erythematosus disseminatus, in only one of which the patient recovered. This patient had a sinus infection, and when the sinus was drained, the skin lesions disappeared and apparently remained absent. Templeton cited a series of cases, in only two of which the patients recovered. Both of these were found to have definite infection around the teeth, and cautious extraction of the teeth, one at a time, with desensitizing doses of autogenous streptococcus vaccine resulted in cure in both instances. Many of the cases due to chronic nasal infection have undoubtedly been overlooked. It is one thing to send a patient to a nose and throat specialist for a check-up, and an entirely different thing to get an adequate check-up. A chronic sinus infection in which transillumination may reveal very little may result in marked toxic absorption due to chronic thickened infected membranes without free pus. Roentgen examination may give negative results because a too penetrating ray was used. If one wishes to get a proper report one must have the cooperation of a nose and throat specialist who is open to reason. I think that all foci of infection should be carefully investigated. We should not be too much puzzled by the cutaneous picture, but should think of the focus that may be back of the picture.

DR. J. FRANK SCHAMBERG, Philadelphia: How conflicting is medical evidence. In this excellent presentation of Dr. Mook and Dr. Weiss the question is asked: If tuberculosis is the cause of disseminate lupus erythematosus, why should we not see more of this condition? Why should we not in the United States see more of lupus vulgaris? How common is lupus vulgaris thought to be in this country? Does it occur in 1 per cent of the cases of tuberculosis? I would venture to say no, without knowing the figures. I was asked by the State Director of Health of Pennsylvania to come up to one of his tuberculosis sanatoriums some years ago and lecture, and among 600 patients with tuberculosis there was not one with lupus vulgaris. There happened to be one case of lupus erythematosus. I doubt if lupus vulgaris occurs in 1 per cent of the cases of tuberculosis, and yet tuberculosis is present in every case of lupus vulgaris. We must be careful in forming conclusions in regard to the frequency of this disease.

I was much interested in this paper for two reasons. The young child to whom I referred a few moments ago with, I believe, fatal erythematosus disseminatus, also had involvement of the joints of serous type, accompanied by a rise in temperature and severe muscular pains. A woman, aged 30, an office patient with a widespread case of lupus disseminatus, had had for three or four years a severe nephritis, with a number of casts in the urine, which necessitated our using a gold preparation in minimum quantities (5 mg.); this had an excellent effect on the eruption. It would disappear for months and then return, and we would then find that the kidneys were again bad. I think that we all appreciate the great difficulties in searching out the etiologic factor in these cases.

Dr. Fred D. Weidman, Philadelphia: Dr. Ayres has already referred to the recent presentation of Dr. Madden. On that occasion I reported a fatal case of lupus erythematosus disseminatus in a woman who had arthritis. At necropsy there were no signs of tuberculosis.

With respect to the ubiquity of tuberculosis, of course, if the tuberculous lesions are present everywhere we should expect to find a large percentage of positive blood cultures, under which circumstances the significance of the high percentage by Löwenstein's technic would be lost, particularly since that technic is such an exact one.

Dr. Charles C. Dennie, Kansas City, Mo.: The matter of disseminate lupus erythematosus will always be a subject for discussion, and I imagine that twenty-five years from now we will still be discussing the etiology of this condition. The fact that a healed lesion of pulmonary tuberculosis cannot be established as a factor and the fact that a patient had active lesions of lupus erythematosus at the time cannot be accepted as a proof, lead me to recite a case in which many of these factors are brought together.

A woman, aged 24, came into the clinic in 1924 with lesions of lupus erythematosus disseminatus all over the body, accompanied with a high temperature. We recognized that the patient was in a serious condition and that treatment of any other kind than expectant would be a serious matter. Through some mistake the internist gave her 0.25 mg. of arsphenamine. Within a short time the lesions became of the Senear-Usher type; in eight weeks they became pustular and all the lesions of lupus erythematosus disseminatus were gone, but the patient developed acute pulmonary lesions. She developed infected glands in the neck, which we aspirated and inoculated into a guinea-pig. Tubercle bacilli were recovered. This woman was sent to Mount Clemens for treatment. I supposed that she had died, but about three months before this meeting I saw her again and at that time she was in good health. A roentgenogram of the lungs showed healed lesions; all the lesions of lupus erythematosus disseminatus were healed, except that they had undergone the changes that many of these lesions undergo, having the characteristics seen when one treats such lesions with roentgen or radium therapy. There were no manifestations of the former disease except the sequelae.

I think that undoubtedly the tubercle bacillus was the etiologic factor, owing to the fact that we recovered this bacillus during the height of the attack, and also from the sputum.

Dr. Oliver S. Ormsby, Chicago: The title of the paper is lupus erythematosus disseminatus, but the discussion has been limited to the acute type. We have all seen a number of cases of disseminated lupus erythematosus of subacute type. Occasionally one of these will develop into, and run the course of, the acute form. Also, occasionally an ordinary discoid type will, through some exposure, develop into the grave type. I saw such a patient who, following a severe sunburn,

developed acute lupus erythematosus disseminatus with lesions that somewhat resembled those of multiform erythema. I am satisfied that some of the fatal cases of multiform erythema are examples of acute lupus erythematosus disseminatus. More often these patients are acutely ill and have marked constitutional symptoms accompanying the cutaneous lesions; these are commonly hospital cases. The disease has exacerbations and partial recurrences, but terminates fatally, as a rule. Of six of these patients whom I have seen within a few years, four are dead, one has recovered and one is now under observation. The Pirquet tuberculin test was made on this patient by another physician and a marked constitutional reaction occurred.

Another case of interest was one that I saw about a year before this meeting. The patient had a high temperature, was very ill and had the usual disseminated cutaneous lesions. For two months she was kept quiet and given sodium thiosulphate twice weekly for the intoxication. It practically cleared the condition up. Then the patient consulted a diagnostic hospital in Chicago to complete the treatment. Here she was given a shock dose of typhoid vaccine which caused a fatal termination in six weeks.

In the subacute cases, unless they develop into the acute type, the patients recover, and I have seen several with extensive lesions always unaccompanied by constitutional symptoms. I have seen eight cases of atypical pemphigus, that is, the group presenting the symptom complex of lupus erythematosus, dermatitis seborrheica and pemphigus, and I am satisfied, on comparing them with cases of lupus erythematosus disseminatus acutus (of which I have several), that they are distinct diseases.

Dr. Walter J. Highman, New York: When I heard this excellent presentation of Dr. Mook's, and when I read his paper in the presessional volume, my comment was "Poor Kaposi!" I was thinking of the paper that Dr. Pusey wrote on the importance of being historically minded. I think that it was forty years ago that Kaposi published his original work. As I recall his views, by lupus erythematosus exanthematicus he meant the acute form that Dr. Ormsby just mentioned, which had nothing to do with the discoid form of lupus erythematosus. The tendency then seemed to be to think that this form might develop in connection with the discoid form of the disease, or that in some exanthematous patients who survived, the discoid type supervened, these lesions having nothing to do with the lupus erythematosus per se.

The exanthematous form of lupus erythematosus, however, is usually a fatal disease. If Kaposi lived today, he would not think that focal infection has much to do with the cases that Dr. Ayres mentioned. Dr. MacKee will undoubtedly recall a case that we had in the Augustan days at the Vanderbilt Clinic. A certain Mrs. Coe had had for years and years what seemed to be a relapsing exudative multiform erythema in the sense of Hebra. By histologic evidence this case turned out to be discoid lupus erythematosus.

Considering these vagaries of the disease, as well as the fugacious relationship of cases of exanthematous lupus erythematosus with the discoid type, and, finally, with the type that we may differentiate as *sui generis* in connection with Dr. Mook's excellent presentation, it seems that we have to admit that acute lupus erythematosus is a disease in its own class, perhaps due to many sorts of infection. The disease is usually fatal, accompanied by general systemic manifestations, such as have been described, which may, even though some are related to lupus erythematosus, be isolated in the group of rare infectious diseases the etiologic factor of which is not yet known. I say this in deference to Dr. Pusey's paper of some years ago on the importance of being historically minded.

DR. JOHN E. LANE, New Haven, Conn.: I wish to refer to one fact that Dr. Schamberg brought up, which has interested me for a long time. It is the relative infrequency of tuberculous skin lesions in tuberculosis sanatoriums. The explanation of this I do not know, but I have corroborated it to my own satisfaction. For fifteen years I think I have seen all the skin lesions in a tuberculosis sanatorium near New Haven, and I think that all the patients with tuberculosis of the skin that have been there were those sent there by me. Neither have I seen a single case of lupus erythematosus there in that time.

DR. JOSEPH GRINDON, St. Louis: The relative infrequency of lupus vulgaris is probably due to the fact that the skin is inherently refractory to tuberculosis, in fortunate contrast to what is true of syphilis. Otherwise, with the ubiquitous character of the tubercle bacillus, one would think that tuberculosis of the skin would be common. Patients with pulmonary tuberculosis do not develop lupus, but patients with lupus develop pulmonary lesions.

DR. WILLIAM H. MOOK, St. Louis: This study of the clinical nature of lupus erythematosus covers a number of years. About two years ago we had our last two or three cases, and we decided to study the material from a clinical standpoint. We approached it with open minds, not prejudiced in favor of or against tuberculosis. We simply took all the evidence that we had and added it up to see what we could get out of it, and there are only two things really that we might emphasize in relation to the study: the leukopenia and the terminal stage of the disease, that is, the serous involvement of the mucous membranes. For that reason we did not think that tuberculosis was the main feature, knowing that practically every one assumes that we all have had tuberculosis at some time. This acute condition did not seem to warrant the tuberculosis being considered the weighing factor in the etiology of the disease.

THE RELATIONSHIP OF DERMATITIS EXFOLIATIVA NEONATORUM RITTER AND IMPETIGO BULLOSA IN THE ADULT *

SAMUEL J. ZAKON, M.D.
CHICAGO

The etiology of dermatitis exfoliativa neonatorum Ritter is as yet not definitely established. In reviewing the literature, one finds the following opinions:

1. Dermatitis exfoliativa neonatorum is but a pathologic expression of the physiologic exfoliation of the new-born (Bohn [1] and Winternitz [2]).

2. Dermatitis exfoliativa neonatorum is closely related to the same group as epidermolysis bullosa hereditaria (Caspary,[3] Elliot [4] and Kaposi [5]).

3. Dermatitis exfoliativa neonatorum and Leiner's erythrodermia desquamativa are one and the same disease (Ronchi [6] and Dalla Favera [7]).

4. Dermatitis exfoliativa neonatorum and pemphigus neonatorum are one and the same disease. The severity and the high mortality of dermatitis exfoliativa neonatorum as compared with the relatively benign pemphigus neonatorum is due to the age of the child (Richter,[8]

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* Submitted for publication, May 15, 1931.

* From the University of Vienna, Clinic of Syphilology and Dermatology. Director Prof. Dr. W. Kerl.

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Hansteen,[9] Hedinger,[10] Hofmann,[11] Delbanco,[12] Pockels,[13] Szarca [14] and others).

5. Dermatitis exfoliativa neonatorum is the cutaneous manifestation of a severe septicemia (Imschenetzky [15]).

6. Dermatitis exfoliativa neonatorum is a malignant form of impetigo contagiosa (Tamm,[16] Gelpke [17] and van der Valk [18]).

Numerous other etiologic factors were suggested at one time or another, such as: scarlet fever (Dorland [19]), syphilis (Prissmann [20]), asphyxia (White [21]), Bacillus pyocyaneus (Guy and Cohen [22]) and pemphigus foliaceus (Behrend [23]).

The following cases meet the clinical postulates of Ritter and present evidence of the close relationship of impetigo contagiosa bullosa and dermatitis exfoliativa neonatorum Ritter.

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REPORT OF CASES [24]

CASE 1.—*History.*—On March 3, 1931, after a normal gestation, a primipara, 22 years old, gave birth to a normal, healthy, female infant. The labor was normal. There were no lacerations, and instruments were not used. The confinement was in the patient's home.

The child progressed normally until the third day after birth, when the mother had the infant's ear lobes perforated. The operation was performed by a midwife, who used a sewing needle. The operation was performed on March 6, and soon the wound became impetiginized. Within the next forty-eight hours erythema and vesiculation appeared, first on the face, then on the neck; and soon the lesions spread from the head downward, becoming generalized on the body. During this time the child did not appear ill, nursed well and had regular bowel movements.

Fig. 1 (case 1).—Appearance of the lesions on admission. The corium is visible and is dry. The epidermis is cigaret paper-like and is removable in large lamellae.

Some days later the clinical picture changed, so that exfoliation took place on all the affected parts. At this time a high fever developed, and the child was brought to the hospital.

Findings and Course While at the Hospital.—The patient was admitted to the hospital on March 12, with a temperature of 39.6 C. (103.2 F.). The epithelium of the cheeks, nose, forearms, legs and thighs was completely denuded. The corium was visible, dark red and dry. On the fingers and toes the epithelium was denuded in glove fashion. On March 13, the temperature taken in the morning was 38.9 C. (102 F.); in the evening, 36 C. (96.8 F.). The child refused to nurse. On March 14, the child died.

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24. These cases were presented at the Vienna Dermatological Society, March 18, 1931, by Dr. E. Urbach.

Necropsy Findings.—The body was that of a relatively large female child for the age (11 days). The epidermis of the entire body, especially, however, of the trunk and extremities, was loose, elevated and removable in large lamellae. The corium was dark red and very dry. The brain showed a slight hyperemia; otherwise, it was normal. The lungs were light, red and filled with air, and the pleura was smooth and shiny. The heart was slightly larger than the fist of the child; the chambers were well closed and contracted. The heart muscle was normal. The foramen ovale permitted the passage of a thin grooved director. The liver was slightly enlarged and of firm consistency. The parenchyma structure was dis-

Fig. 2 (case 1).—Epithelium denuded in glove fashion. Note the rolled up edges.

cernible. The spleen was normal. Both suprarenal glands were markedly enlarged and rich in parenchyma. The kidneys were normal. The gallbladder protruded from beneath the lower edge of the liver and was filled with clear, stranded, mucous fluid. The bile passages were normal. The stomach and intestines were normal as to both contents and wall structure.

Microscopically, the tubular epithelium of the kidney showed minimal cloudy swelling. The liver cells showed vacuolization and stasis.

Sections from the affected portions of the skin showed edema and hyperemia of the corium and the papillary body, though the cellular exudation was minimal.

In several places the epithelium was missing, and the surface of the corium was covered with a fibrinous exudate. The epidermis showed in some places absence of stratum corneum, but in several places the corneum was parakeratotic and lifted up from the underlying structure.

This case is of interest because the mother, the father and the aunt of the child, through coming in contact with the child, developed lesions of an impetigo bullosa type.

Case 2.—*History of the Mother.*—On March 3, 1931, the patient gave birth to a healthy child. The gestation and labor were normal. On March 6, blisters and redness developed all over the body of the child following impetiginization of the ear lobes. On March 9, the mother noticed that large bullae had appeared on the inner part of the upper third of her thighs.

Status.—At the time of the patient's admission to the clinic on March 12, 1931, there were numerous flaccid bullae on the inner side of the upper third of the thighs; underneath the vulva were many blisters. The contents of the blisters was almost clear. The skin surrounding the blisters was not reddened. On first appearance, the lesions gave the impression of pemphigus-like bullae.

Clinical Course.—On March 13, the temperature was 37.5 C. (99.5 F.). The blisters were healing. On March 15, many new blisters appeared on the face. After they were opened, red spots remained for days. On March 18, a large coin-sized blister appeared over the left nipple. On March 19, the patient suffered from severe mastitis. She was referred to the obstetric clinic, where she made an uneventful recovery.

Other Relatives Affected.—On March 9, the child's aunt, a 20 year old girl, who also had come in contact with the child, developed on the right index finger a bulla the size of a lentil. A culture made of serum from this bulla showed a rich growth of hemolytic streptococci.

On March 11, blisters developed on the bearded parts of the face of the father of the child.

CONCLUSIONS

Cases are reported that show, as many other cases described in recent years have demonstrated (Gelpke,[17] Tamm,[16] van der Valk[18]), that dermatitis exfoliativa neonatorum Ritter (case 1) and impetigo bullosa of adults (case 2) are probably the same disease, and that the difference in the clinical appearance and course in both conditions is due to the differences in the biology of the skin of an infant and of an adult. Therefore, one may suppose that dermatitis exfoliativa neonatorum Ritter, impetigo bullosa and pemphigus neonatorum are all forms of impetigo contagiosa bullosa.

EARLY EPITHELIOMA*

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MANCHESTER, ENGLAND

CASE 1.—An elderly woman was referred to me by her physician because she had a warty growth on the right cheek (fig. 1). The tumor was of three months' duration. It presented a central protuberant keratotic mass set in a fleshy base, and suggested the appearance of an early cutaneous horn. The growth was freely movable with the skin and but slightly infiltrated it. There was no evidence of infection or inflammation. No adenitis could be appreciated. In spite of the apparently long duration of the tumor and the absence of lymphatic invasion, I had no doubt but that we were dealing with a slow-growing, squamous carcinoma (epithelioma). The biopsy confirmed my diagnosis. Further examination of the patient's face revealed on her forehead, and partly covered by her hair, a little tumor, of a week's duration, which was the color of the surrounding skin and slightly raised above its surface. There was a mild central keratosis, so that at a little distance the appearance of the lesion suggested a plugged follicle, although closer investigation showed that such a condition did not exist. There was no evidence of infection or inflammation, and no substance could be expressed by squeezing. It was difficult to determine the amount of infiltration of the skin beneath the lesion. Had I seen this tumor twenty years before, I might have hesitated in pronouncing such a small, benign-looking excrescence an epithelioma; but, having seen many of these early carcinomas, I had no hesitation in making a definite diagnosis of squamous carinoma, and figure 2 confirms my opinion.

Early lesions such as this one are not usually referred to a physician, but are discovered more or less accidentally or, as in this case, by thorough examination of a patient who has at the time another well established lesion. When in a case of squamous carcinoma of the face another carcinoma appears later elsewhere on the face, it is usually of the same type of growth as the first tumor. In the case under consideration, however, though I found that to be true, I also found scars on the bridge of the nose and on the left cheek, which were the sites of former basal carcinomas (rodent ulcers) for which I had treated the patient previously. Further examination revealed also, at the sides of the forehead and in the malar regions, small keratotic lesions that are usually spoken of as senile keratoses. It is well recognized that in these cases of senile keratoses multiple carcinomas, of either the squamous or basal type, are prone to appear, but in my opinion the keratotic lesions are the first clinical manifestation of a slow-growing carcinomatous condition of the skin, and the tumor that is evident later is not a supervention but simply a progressive development of the already existing intra-epidermal carcinoma.

* Submitted for publication, March 14, 1931.

Keratosis is the first clinical evidence of a squamous carcinoma of the skin. The following case (fig. 3) I think supports my contention.

CASE 2.—The patient was a man, aged 80, whom I had seen from time to time during the past twenty years, and from whose face I had removed many carcinomas, both squamous and basal. On the left cheek was a scar, the result

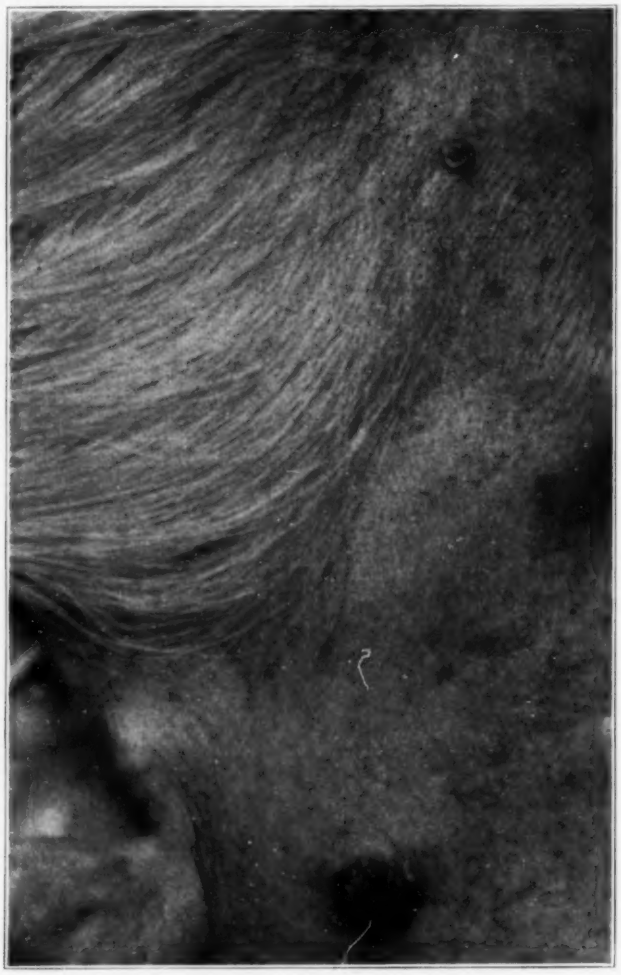

Fig. 1 (case 1).—Early epitheliomas in a case of senile keratosis.

of the excision of an epithelioma forty years ago. To the outer side of the right external canthus was a squamous carcinoma of twelve months' duration and in the center of the tumor there could still be seen a small cap of keratosis beneath which the growth developed. On the nose and cheeks were atrophic areas, the sites of former basal carcinomas, and scattered all over the face were numerous small keratotic lesions. I had recently excised one such lesion, in front of the right

Fig. 2 (case 2).—Photomicrograph showing early epithelioma of one week's duration.

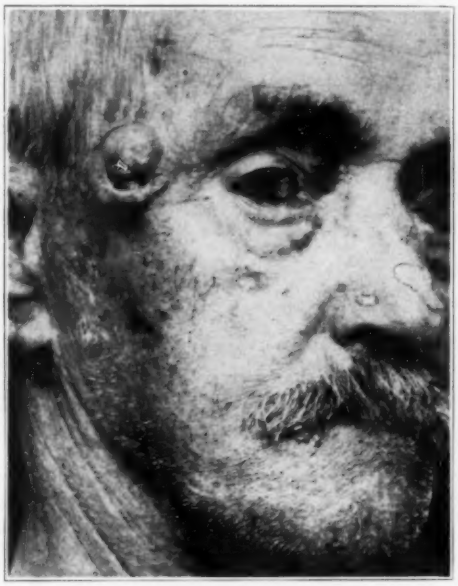

Fig. 3 (case 3).—Early squamous and basal carcinomas in a case of senile keratosis.

ear, not more than one third of an inch (0.8 cm.) in its longest diameter. Figure 4 shows the histologic picture. In the small area portrayed there was evidence of three distinct and separate growths. In the center was a typical basal carcinoma, on the right an early (intra-epidermal) squamous carcinoma and on the left a small nevus.

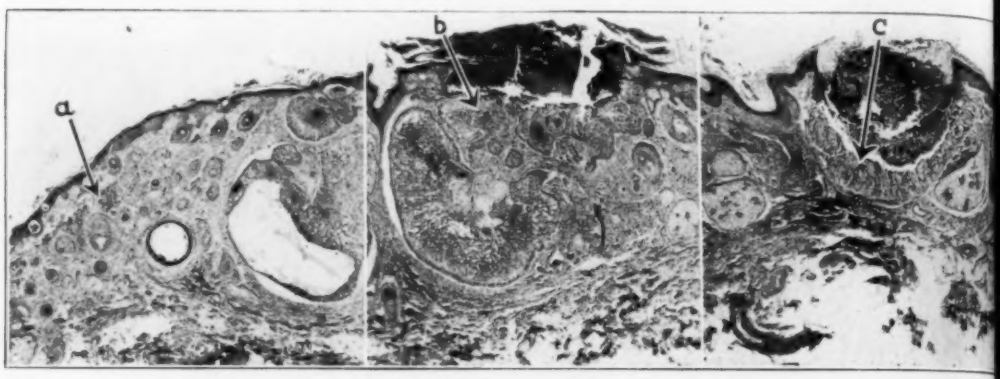

Fig. 4 (case 2).—Photomicrograph showing growths in a case of senile keratosis: *A*, a small nevus; *B*, a basal carcinoma, and *C*, a squamous carcinoma.

Fig. 5.—Early squamous carcinomas of the backs of the hands in a roentgen ray operator.

The second patient had spent all his life on inland waterways; so his condition might be considered as coming under the category of Unna's sailors' skin. The first carcinoma was removed when he was 40 years of age. The first patient, however, had always had a sheltered

and sedentary occupation. That the actinic solar rays are carcinogenic in predisposed skins I have little doubt, but this realization does not leave us much nearer to the determination of the cause of cancer. The carcinogenic effects of roentgen rays are also well known. Figure 5 illustrates "warts" of the hands in an operator who had worked with roentgen rays for twenty-five years. The picture presents multiple slow-growing squamous carcinomas of the skin of the hands. Those represented by *A* and *B* are, to my mind, palpable epitheliomas; histo-

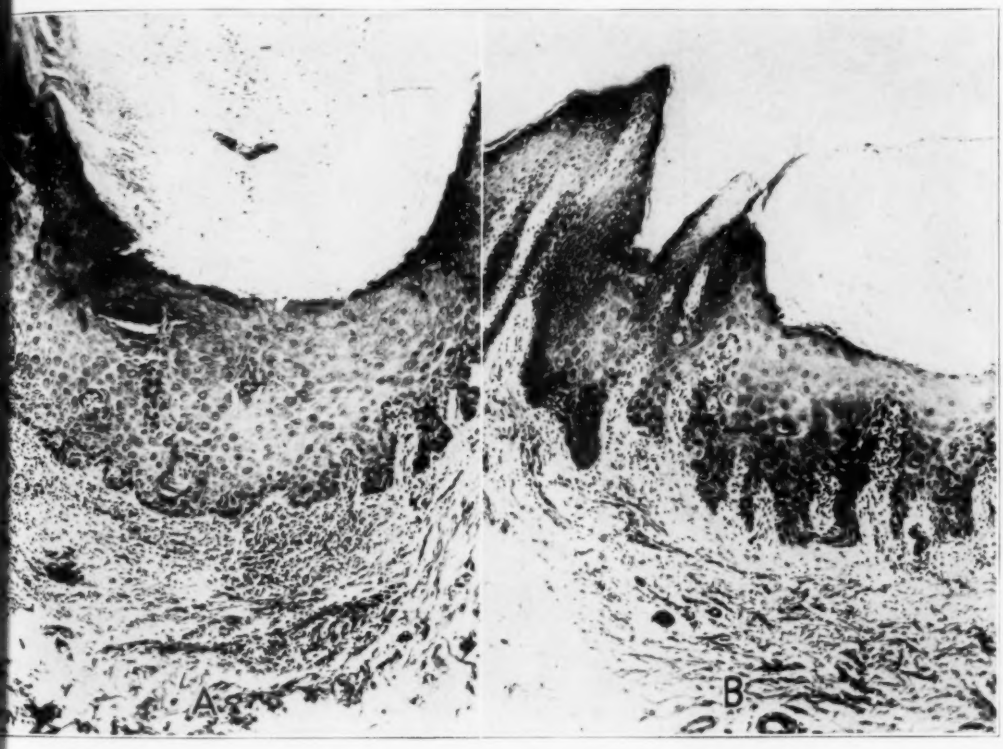

Fig. 6.—Photomicrographs showing the early stages of a small early squamous carcinoma (fig. 5, *C*). *A* shows the intra-epidermal stage, and *B*, the second stage of the development of the carcinoma, including the budding process.

logic examination of the smaller lesions will demonstrate their carcinomatous nature. One of these (*C*), one eighth of an inch (0.3 cm.) in diameter, was excised, and figure 7 shows the invasion of the dermis by the proliferating epidermal cells. Figure 6 shows the earlier (intra-epidermal) stage of this growth.

We are frequently told that epitheliomas develop on warts and papillomas of the skin. Under the latter term are included all sorts of excrescences. The fact is that the early epithelioma is often mistaken

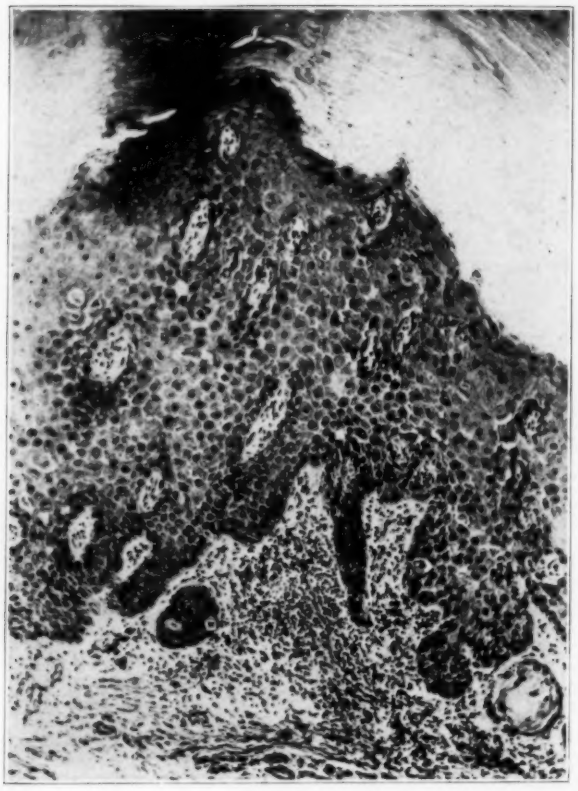

Fig. 7.—Photomicrograph showing the stage of invasion of the dermis in a small early squamous carcinoma (fig. 5, *C*). Note the development of "cell nests."

Fig. 8.—Early epithelioma on the back of the hand.

for a wart or papilloma. The so-called senile warts on the back of the hands of elderly folk are slow-growing squamous carcinomas. Figure 8 illustrates a typical case. The tumor was of twelve months' duration, and there was no invasion of the lymphatics. It was presented to me as a persistent wart, and had it been allowed to grow for another twelve months I should probably have found a more pronounced growth with lymphatic invasion and with the history that the "cancer had developed on a wart." The more precancerous lesions are investigated the fewer are found.

I have stressed the points made because the student finds so little in books on dermatology to guide him in the diagnosis of early epitheliomas of the skin.

TUBERCULIDE EN PLAQUE*

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Tuberculids appearing in the form of plaques are rare and are a type not usually mentioned in the descriptions of tuberculosis of the skin.

Hauser,[1] in 1920, reported two cases of large papular and annular tuberculids. His case showed no signs of suppuration or later necrosis and did not fit into the usual group of tuberculids. Hauser gave them a special designation, "Large, papular, grouped and plaque formed tuberculide." He stated that such forms have rarely been mentioned in the literature.

J. Jadassohn [2] mentioned a grouped, large, papular tuberculid in his paper before the Eleventh German Dermatological Congress in Vienna, in 1913.

Hauser was of the opinion that the microscopic picture depended on differences in the immunity reactions of the tissues. Different pathologic conditions can be caused by tubercle bacilli coming by way of the blood stream and lodging in the skin. An inflammatory necrotic process causes a marked reaction of the tissues with rapid destruction of the bacilli.

All tuberculids are a form of hematogenous tuberculosis brought about in a similar way, and the clinical differences are caused by various factors, such as number of bacilli, virulence of the organisms and the immunity reactions of the tissue, so that it is not necessary to separate the large papular tuberculid into a separate form of tuberculosis.

The case reported here was presented before the Minnesota Dermatological Association on Oct. 6, 1926.[3]

Histologically, it showed the picture of tuberculosis, but clinically it did not resemble any previously published descriptions of that disease that I have seen.

Prof. Oscar Gans of Heidelberg, who was present at the meeting, offered a diagnosis of tuberculide en plaque, a designation that seemed to cover the description of the case perfectly.

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* Submitted for publication, Feb. 8, 1931.

* Read at the Fifty-Fourth Annual Meeting of the American Dermatological Association, Toronto, Canada, June, 1931.

* From the Division of Dermatology, University of Minnesota.

1. Hauser, A.: Arch. f. Dermat. u. Syph. **128**:149, 1920.

2. Jadassohn, J.: Arch. f. Dermat. u. Syph. **119**:10, 1914.

3. Sweitzer, S. E.: A Case for Diagnosis, Arch. Dermat. & Syph. **15**:354 (March) 1927.

This patient has been observed for four years, so that it is possible to report on the appearance of the lesion at a later date as well as in 1926.

REPORT OF A CASE

History.—One and one-half years before examination, the mother of M. P., a girl, aged 12, noticed a small reddish area on the anterior surface of the child's right ankle. This spot became larger and turned to a bluish area much like a black and blue mark. Since then spots had appeared on both lower extremities, and lately on the arms and wrists.

Fig. 1.—Plaques as they appeared when first seen in 1926.

Since the beginning of the condition the patient had been more or less inactive and had complained of being worn out. No rise in temperature had been noticed, but on several occasions the mother thought the child was feverish.

The past history and the family history were unimportant, except that a maternal uncle died of tuberculosis. The general physical examination gave negative results. The lesions of the skin varied in size from 1 to 5 cm. in diameter; some were round and others oval, and in places gyrate figures were formed. The lesions varied from purplish to brownish in the center and were lighter purple at the periphery. There was an apparent network of minute capillaries over the larger lesions. The earliest lesions were small macules about

the size of a pea. There was no exudation or scaling or any evidences of scratching. The larger lesions were firm and raised above the level of the skin, forming flat, hard plaques. They were located on the anterior and posterior surfaces of the lower extremities, beginning at the ankle, and a few lesions were present on the arms and in the axilla. There was some early eruption on the lower portion of the abdomen. The face, scalp and neck were free.

Examination and Course.—The patient was admitted to the dermatologic wards of the General Hospital for observation. During her stay of a few days

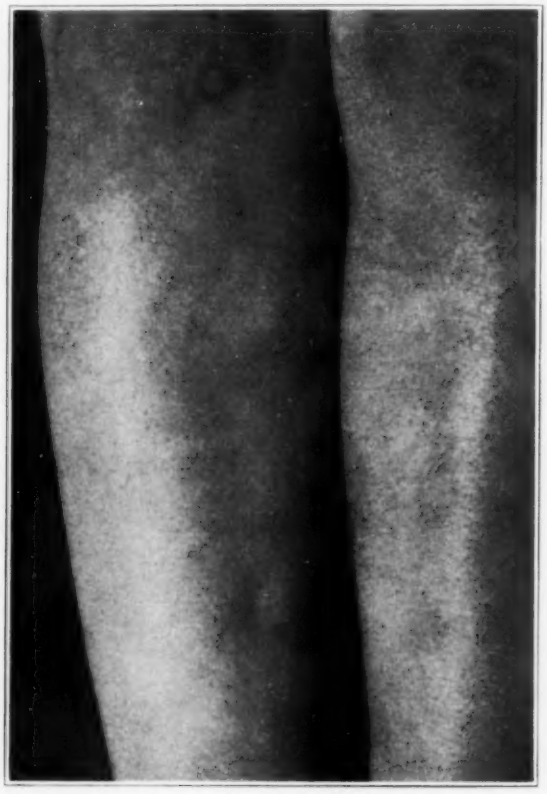

Fig. 2.—Depressed scars of healed lesions after treatment.

the temperature was 99.2 F. on only one occasion. Examination of the urine and blood gave negative results. The Wassermann, Moro, Pirquet and subcutaneous tuberculin tests were negative. X-ray plates of the lungs showed an increase in the hilar shadows but no definite evidences of tuberculosis. A biopsy was made, and material was inoculated into a guinea-pig. Six weeks after inoculation, a postmortem examination of the guinea-pig did not reveal tuberculosis.

The patient was given arsenic in the form of neoarsphenamine, combined with sunlamp treatments, and in the course of a year the lesions disappeared, leaving at the site of the larger lesions soft, smooth depressed scars, greatly resembling the scars left by Boeck's sarcoid.

Fig. 3.—Recurrence in 1930; elevated plaques on ankle.

Fig. 4.—Plaque on wrist with an outline resembling granuloma annulare.

Fig. 5.—Close view of a plaque.

Fig. 6.—Low power magnification showing dense infiltrate in the cutis.

The patient was examined regularly in 1927, 1928 and 1929 and presented for the most part only small groups of faint, new macules along with the depressed scars of the former plaques.

In 1930, the groups of new macules became more prominent and gradually formed plaques over the dorsa of the feet and at the side of the left wrist. These plaques were firm, elevated and yellowish brown and looked the same as the plaques seen in 1926. The tuberculin, Moro and Pirquet tests at this time were again negative.

Histology.—Histologic examinations were made from a number of lesions removed at various stages of evolution. The pathologic changes were essentially

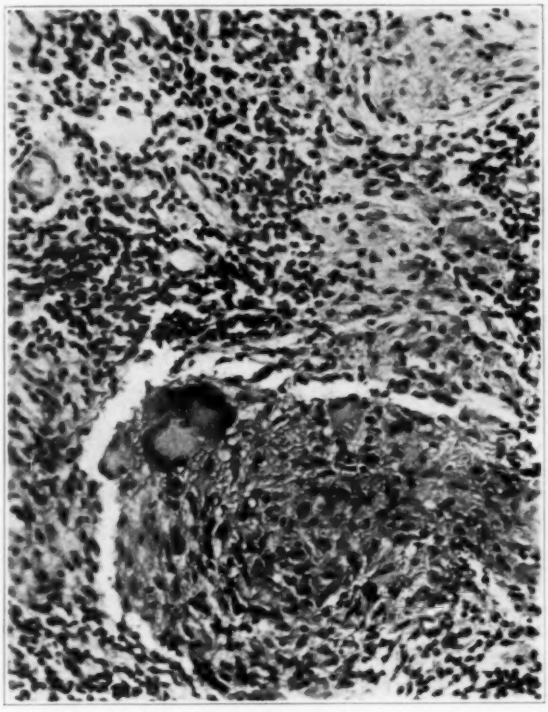

Fig. 7.—High power magnification showing details of the tuberculoid structure.

the same. There was no primary epidermal change, only a degree of atrophy and slight scaling, which undoubtedly was secondary to the dense infiltrate. The striking feature of all the sections was a marked infiltrate of the papillary and subpapillary portions of the cutis. This infiltrate was made up of two distinct types. In zones there were areas of inflammation made up entirely of lymphocytic cells. Adjacent to this there was a closely packed infiltrate of epithelioid cells, round cells and an occasional giant cell. Close search of the corium revealed here and there areas that closely resembled true tubercles, but, as was to be expected, there was no necrosis. A marked perivascular infiltrate was also found in spots, and giant cells were located along small blood vessels. Elastic tissue was missing in the infiltrated zone, and there seemed to be no particular areas about the central

blood vessels. Sections stained for tubercle bacilli revealed no acid-fast organisms. The conclusion from the examination of the section would be indurative tuberculosis (H. E. Michelson) without demonstration of the bacilli.

COMMENT

Where should this interesting case be placed? The histopathology showed a tuberculoid structure, but it is well known that tuberculoid reactions are not specific for tuberculosis and are found in many diseases such as blastomycosis, trichophytosis, syphilis, leprosy and sporotrichosis.

Professor Gans, after a careful study of the patient and the sections of skin, was inclined to favor a diagnosis of tuberculosis, and suggested the name tuberculide en plaque.

Besides tuberculosis, erythema elevatum diutinum, granuloma annulare and sarcoid were considered.

Crocker and Williams,[4] in 1894, published an article on erythema elevatum diutinum. They described nodules varying in size from that of a small pea to that of a bean, pink in the early stage and purplish when of long standing. The lesions were convex at first; they tended to coalesce into irregular, lobed, infiltrated and flat, raised plaques, but in severe cases distinct nodular tumors were present, even on the palms and soles.

The growths are firm to the touch and painless. They develop on the extensor aspect of the limbs over the articulations, elbows, knees and phalanges of the hands and feet. They also affect the palms and soles, buttocks and ears. They may involute or may persist for years.

All but one of the cases noted by Crocker occurred in female patients and all were in children or young adults. Gout or acute rheumatism either was present in the patients or was mentioned in their family history.

The histopathology showed the lesion to be located below the epidermis in the deep portion of the corium. It consisted of a fibrocellular structure which in a great measure replaced the normal fibers of the corium. The fibers followed the course of the vessels.

Trimble[5] considered erythema elevatum diutinum an entity and not a variant of granuloma annulare, as granuloma annulare is a ringed eruption of small nodules arranged in a circle or segments of a circle with a normal or atrophic center, while erythema elevatum diutinum is a raised, flat, pink or purplish plaque without a central clearing.

According to Hartzell,[6] the main histologic feature of granuloma annulare is a deep infiltration with an area of central necrosis. The

4. Crocker and Williams: Brit. J. Dermat. **6**:1, 1894.

5. Trimble, W. B.: Erythema Elevatum Diutinum: Report of a Case, with Comments on Its Nosologic Position, Arch. Dermat. & Syph. **13**:383 (March) 1926.

6. Hartzell, M. B.: Tr. Sect. Dermat. & Syph., J. A. M. A., 1914, p. 27.

histopathology of erythema elevatum diutinum is one of fibrosis. Hartzell thought that these two conditions were the same; Trimble thought the opposite.

Weidman and Besancon,[7] in 1929, reported two cases of erythema elevatum diutinum, both of which were examined histologically, and in one of which they obtained a pure culture of Streptococcus ignavus. The histology of both cases was identical and showed acanthosis in the epiderm and an acute periarteritis and peritelangiitis in the corium. The cells in the infiltration were predominantly polymorphonuclears. Weidman and Besancon looked on the condition as rheumatic in origin and considered it an entity distinct from granuloma annulare.

Rost,[8] in his excellent monograph on tuberculosis of the skin, placed granuloma annulare in close connection with tuberculosis and called attention to the fact that Dittrich [9] demonstrated the presence of acid-fast bacilli in this disease.

The patient in the case reported here showed some features that were to be compared with the eruption of granuloma annulare. In this patient the lesions did not tend to make such distinct rings or crescents, and the color was red or blue, never the ivory white of granuloma annulare. The location of the involvement was in areas that were rarely invaded by granuloma annulare.

Both from a clinical standpoint and from the histologic signs, it was possible to rule out a diagnosis of erythema elevatum diutinum and of granuloma annulare.

Nosologically, this case seems to me to be closely related to the indurative types of tuberculosis. This view is substantiated when one chooses certain areas in the histologic section that have a striking resemblance to sarcoid, and in other places the structure is that of erythema induratum. Viewing the section as a whole, there is an absence of lobulation as seen in sarcoid, and the infiltrate is not so distinctly perivascular or so deeply situated as in Bazin's disease.

Goldsmith [10] recently reported a case in which the clinical diagnosis was morphea, but histologic examination revealed changes that had not the remotest resemblance to those hitherto described in that disease. The patient was a middle-aged married woman with irregular brownish-yellow patches, hard and smooth on the lower part of the legs. The Wassermann test was negative, the tuberculin reaction was moderately

7. Weidman, Fred D., and Besancon, John H.: Erythema Elevatum Diutinum: Rôle of Streptococci, and Relationship to Other Rheumatic Dermatosis, Arch. Dermat. & Syph. **20**:593 (Nov.) 1929.

8. Rost, George A.: Diagnose und Therapie der Hauttuberkulose in der Praxis, Leipzig, Johann Ambrosius Barth, 1930, p. 57.

9. Dittrich, O.: Arch. f. Dermat. u. Syph. **153**:599, 1927.

10. Goldsmith, W. N.: Brit. J. Dermat. **41**:226 (June) 1929.

positive. Histologically, the sections showed a tuberculous structure, a granuloma with numerous giant cells.

Inoculation into a guinea-pig gave a negative reaction. Goldsmith concluded that one must face the possibility that tuberculosis can give rise to a scleroderma-like manifestation.

This case reported by Goldsmith bore a close resemblance in some particulars to my case. Recently MacCormac [11] reported a case of tuberculid of the morphea type, which he said resembled Goldsmith's case.

Gougert and Burnier,[12] in 1929, reported a case of sclerodermiform sarcoid of the lower extremities which had some points of resemblance to my case. They considered the condition in their case as a transition type of tuberculosis occupying a place between the sarcoid of Boeck and the sarcoid of Darier-Roussy. The difference histologically between their case and my case was that giant cells and epithelioid cells were found in the latter.

CONCLUSIONS

A case is reported of a girl, aged 12, with numerous hard elevated plaques on the lower extremities. These healed in about a year, with the formation of smooth, soft, depressed scars. Numerous groups of macules and papules were also present on the upper part of the limbs, abdomen and arms.

All tests were negative, including inoculations into guinea-pigs. In spite of these negative observations, the histology was that of tuberculosis. Clinically, the condition did not fit into any of the known diseases of the skin. Therefore, from the microscopic picture and clinically, by exclusion, a diagnosis of tuberculide en plaque was made.

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ABSTRACT OF DISCUSSION

DR. H. E. MICHELSON, Minneapolis: Since this is the only paper on the program that deals with tuberculosis of the skin, I feel justified in taking up the subject in a more or less general way.

I believe that the time will come when we will discard most of the present terms that are applied to tuberculosis of the skin. At present we have some striking names which are important because they signify the location of the lesion. For example, lupus pernio implies a definite entity clinically, and still, microscopically, it might well be called sarcoid. There must be a definite connection between the manifestation and the classification, but there are no tangible laws as far as we know that regulate the histologic changes. A tuberculid shows certain signs at one time while a specimen taken for biopsy a few days later may look entirely different. Evidently there is a battle going on between the structures and the invader. Professor Kren has determined that tuberculosis of

11. MacCormac, H.: Proc. Roy. Soc. Med. **23**:1031, 1930.

12. Gougerot, H., and Burnier: Arch. de dermat. et syph. de la clin. de l'Hôp. Saint Louis **1**:155, 1929.

the skin is always brought about by bacillary emboli, and believes that one might use a terminology that covers all tuberculosis of the skin, using a modifying adjective to signify location. Surely the most important advance has been the positive culture in tuberculosis, and the future criteria for diagnosis will probably be the culture, and not the histologic findings.

DR. WALTER J. HIGHMAN, New York: For a great many years I have been interested in the relationship of the tuberculids to tuberculosis in the analogous sense to the relationship of tricophytids to tricophytosis, a relationship that in a large part tended to be clarified by the work of Peck, Sulzberger and Bloch. Peck and Bloch showed that the tricophytid is due to a phytemia in which the Tricophyton in an attenuated form has produced a lesion that resembles the lesion of true infection, but differs from it since it contains no demonstrable organisms. It seems to me that the tuberculids and tuberculosis must be analogous.

The case Dr. Sweitzer has presented is interesting and instructive from the standpoint that the tuberculous lesion, the tuberculid, the intrinsic form of tuberculosis and granuloma annulare are all simulated, if not actually reproduced. It seems to me that probably in their great work Lewandowsky and Jadassohn discussed these variegated forms of tuberculids, actually representing the metastatic, the embolic, the allergic reactions of the nonviable tubercle bacillus, or the tuberculous toxin, in the skin, creating lesions that are in effect similar to tuberculosis both clinically and histologically, without, however, the tubercle bacillus remaining capable of growth on proper culture mediums, if parasites indeed be demonstrable by such proof as we can obtain through animal inoculations or otherwise.

It seems to me that Dr. Sweitzer's case is significant far beyond its multiple clinical manifestations, in so far as we can indicate when we talk of tuberculid, if you will, or tuberculosis en plaque, what the tubercle bacillus may produce in the skin in a nonbacterial sense, using the term bacterial in the sense that the tubercle bacillus is viable. I fancy that this particular case, through the multiformity of the lesions and the multiple histologic manifestations, which Dr. Sweitzer has chosen to designate as tuberculid en plaque, is really rich in biologic meaning.

DR. LLOYD W. KETRON, Baltimore: I should like to speak briefly in this connection concerning the difficulty of proving the presence of tubercle bacilli in the majority of instances in which we make a diagnosis of tuberculosis cutis. There have been, perhaps, more cases, lupus excepted, diagnosed tuberculosis of the skin in which the organism could not be demonstrated than in which its etiologic relationship has been definitely proved. For the past two or three years we have been inoculating animals, mainly guinea-pigs, in quite large numbers from several types of so-called skin tuberculosis by a method of successive transfer which has been advocated as giving positive results much more frequently than the usual procedure. By this method the ground up material is injected into the inguinal region of the animals, and after several weeks the glands are removed and reinjected into a second lot of animals and so on until two or three transfers are made. So far we have had only one positive result which was from material taken from the only case of lupus with which we worked. In the others, including some forms of tuberculids, lupus miliaris disseminatus, erythema induratum and a few other cases which were not definitely placed in any of the common groups, our results were entirely negative. It is quite possible to believe that pathologic changes in the skin may be produced by the tubercle bacillus which may be destroyed later by local defensive mechanisms, and that the lesions may persist for a considerable length of time owing to the presence of the organisms in the

tissues, although they are dead. It is hard to see, however, how this can be the case when such lesions are increasing in extent. Our investigations have made us wonder whether or not we are not going a little too far in diagnosing tuberculosis of the skin based only on microscopic pathology and clinical appearance.

DR. CHARLES C. DENNIE, Kansas City, Mo.: There is just one point I wish to mention, that has been observed before. The technic of inoculation of guinea-pigs with supposed tuberculous lesions is important. We were getting far too many positive reactions in our inoculations, and on investigation found that the old man who was taking care of the pigs had tuberculosis. I think we should always examine the caretakers, for if they happen to have tuberculosis the guinea-pigs are very likely to become contaminated.

DR. ARTHUR W. STILLIANS, Chicago: I wish to bring to your attention in connection with Dr. Sweitzer's report a case that we presented before the Chicago Dermatological Society in 1926. The patient was a Negress, aged 26, whose lesions had been present for three years on the face, the posterior aspect of the arms, thighs and legs. Many of these plaques had at the center translucent brown papules, which persisted. Pulmonary findings were not typical of tuberculosis but suggested it. There were mottling in the lower part of the lung and interlobar pleurisy. The pulmonary condition grew worse during the patient's pregnancy, but she passed through the pregnancy all right. The lesions persisted all the time we were in touch with her, but as she disappeared from observation we do not know whether or not they cleared up. We reported the case at that time as sarcoid because of the histologic appearance, but we were never satisfied with that diagnosis.

DR. LLOYD W. KETRON, Baltimore: With reference to animal inoculation, the mistake is usually made that the animal is killed after six weeks. Of course, if it has already contracted tuberculosis it is of no importance, but a negative report is certainly not conclusive, for it occasionally takes three or four months for apparent infection to take place. Furthermore, more than one animal should be used, and it should be carefully tested with tuberculin before inoculation to rule out the possibility of an infection in the laboratory.

DR. J. FRANK SCHAMBERG, Philadelphia: I only wish to state that during twelve years we have never had a case of spontaneous tuberculosis in a guinea-pig.

DR. SAMUEL SWEITZER, Minneapolis: I only wish to emphasize that in this case the inoculation into the guinea-pig was negative when the pig was killed in six weeks, so this test was probably no good.

I also wish to stress the point that the blood cultures, if they are worked out by the method of Kren, will probably give a better diagnostic means than any we have had so far. Kren and his co-workers claim that their method is far superior to any other we have had. We tried the culture, but evidently we did not have the proper technic as our results in this case were negative and cultures from active pulmonary tuberculosis were negative.

DERMATOLOGY IN SOVIET RUSSIA*

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At the conclusion of the recent International Congress in Copenhagen, I made a short visit to Russia to get a glimpse of that strange country and to learn what I could about the present status of dermatology. Contrary to the prevalent idea, it was not at all difficult for an American to either enter or leave the country. I had enough letters of introduction to satisfy the most exacting official, but had no occasion to use the majority of them. These precious documents included a letter from Senator Borah, a name to conjure with in Russia, a letter from Surgeon-General Cumming, and one from a dermatologic colleague, Dr. M. Michailovsky of New York, whose advice proved to be invaluable.

The necessary plans for my visit were made at home through a representative of the so-called "Intourist" bureau of Russia. All that remained was to obtain a visa in some country that had officially recognized the Soviet Government. This was done at the Russian consulate in London, though not with Yankee celerity. Crossing the Russian frontier from Finland was a simple matter, though all foreign currency had to be changed at the border to Russian money. Any amount of money in the form of letters of credit and travelers' checks was admitted freely. I might add that a reasonable amount is needed, as even mediocre accommodations in Russia at present are expensive. Riding in a dilapidated droschky from the depot to my hotel in Leningrad cost five rubles ($2.50). It was worth the price, however, to be driven by a picturesque whiskered person who looked the part of a pirate. He may, however, have been a most exemplary citizen, for at least he took good care of his horse, which in the excellence of its appearance was in marked contrast to man and vehicle.

Leningrad looked rather shabby and deserted in contrast with Moscow, which is seething with life and in which many new buildings are being constructed. All of the men I saw looked rather spare, whereas the women appeared to be much more sturdy. At any rate, during my brief visit in Russia I did not see any one who needed "reducing." Most of the men wore caps and many of them characteristic Russian blouses, including Dr. Vladimirsky, the Commissioner of Public Health. This particular gentleman, like many others from the

* Submitted for publication, March 21, 1931.

old régime, had been educated in Paris and spoke French fluently. Like all of the medical men I met, he was most courteous and kind.

I was astonished at the large number of persons on the streets and in the recreation parks of Moscow. This was partly due to their system of working in shifts, each man or woman spending four days at work and resting on the fifth. There was consequently nothing to correspond with the Saturday afternoon and Sunday of the United States, when the majority of the citizens, except doctors and a few others, take a holiday.

For one whose time is short and who wishes to visit numerous hospitals it is advisable to have a guide, though this is not essential. Practically all the guides are students in the universities, mostly young girls who are studying various languages. My guides in both Leningrad and Moscow were intelligent girls about 20 years of age, who were anxious to point out the accomplishments of the Soviet régime. Their ideas about America were somewhat distorted, one of them asking me whether it was not true that thousands of people in America were starving to death every day in spite of the great wealth of the country. I did my best to disabuse her mind of any such monstrous idea.

In the course of my visit I talked with a number of American and British business men; I asked innumerable questions of my guides, and went with them to as many movie shows and theaters as time would allow. I did not have an opportunity to become well enough acquainted with any Russian physicians to learn their opinions regarding the type of state medicine that prevails in Russia.

As I am not an economist and as my visit was brief and superficial, it is not my place to discuss the economic or political aspect of Soviet Russia. In regard to the status of dermatology it was difficult to estimate what advances had been made by the present government as I had no means of comparison with the former régime, this being my first trip to Russia. I am convinced, however, that the Soviet Government fully appreciates the importance of good health and does everything in its power, not only to take care of those who are ill, but also by means of radio and moving pictures to disseminate information concerning public health and personal hygiene. The propaganda against all forms of religion is well known, although, as far as I can learn, there is at present no religious persecution. The religious fervor of the Russians is now directed toward the colossal plan of industrializing their country and, among other things, safeguarding the health of the people.

The famous linguistic talent of educated Russians was not particularly apparent. All of the older physicians whom I met could speak one or more foreign languages. On the other hand, I did not meet a single doctor who had graduated since the revolution who could speak any language but Russian. In their fierce struggle for existence time had evidently been lacking to develop their national talent for languages.

Fig. 1.—Bronner Institute for Venereal Diseases (Moscow).

Fig. 2.—Lecture to patients in a waiting room at the clinic.

In talking with this group my guides were indispensable, for my knowledge of Russian is extremely limited.

While I visited some of the leading clinics and hospitals for cutaneous diseases in Leningrad and Moscow, August was a poor time to do so, as many of the physicians were absent on their vacations, and some of the clinics were closed for much needed renovation. Only a few Russians were allowed to leave the country on business or professional trips. For instance, the delegation at the Congress in Copenhagen consisted of only two men, Professor Meschersky, holding the chair of dermatology in the First University at Moscow, and Dr. Bremener, in charge of the Light Institute in the same city. This was indeed a small number from such a vast country.

A word about women doctors and medical titles in Russia might be of interest. It is well known from many authoritative books of recent date that women have been completely emancipated in Russia, and that they not only have full suffrage, but fill many important positions in the government. I was interested to learn on my visit to the Tarnowsky Hospital that the director of a large service was a woman, Prof. Olga Podvisotskaya. In fact, there were two women in Leningrad who held this rank. While the medical titles of professor or privat-docent are naturally held in esteem, no one in Russia, not even Stalin, the head of the government, is called by any other title than Comrade or Citizen. Foreigners are called by their proper titles through courtesy.

In my visits to various medical institutions in Leningrad and Moscow I took notes and asked many questions, but was unable to obtain more than a part of the information I desired. In consequence, I gave Prof. V. M. Bronner of Moscow a list of questions of a statistical nature, which he kindly answered in full a few months after my departure. The data that follow are therefore based partly on personal observation and partly on information given by Professor Bronner.

The general scheme of medical education in Soviet Russia differs in some respects from that in other countries of Europe and America. The medical colleges of Russia now include the following three faculties: first, "Therapeutic-Prophylactic," which is divided into the departments of therapeutics, surgery and stomatology, training the student to become a physician, surgeon or dentist; second, "Sanitary-Prophylactic," which teaches the general principles of public health and sanitation and also trains specialists in industrial sanitation, food supply, epidemiology, etc.; third, "Prenatal Care and Child Hygiene."

The periods of study with the first and third faculties are four years, each year consisting of 320 working days. The department of stomatology, however, requires only three and one-half years. At the conclusion of the four year course students are required to serve an additional year in some hospital before obtaining the right to do independent medical work. I was unable to obtain an answer to my question as to whether

physicians in Russia may engage in private practice or whether they are all employed by the state. This was the only one of my questions that was not answered directly and explicitly. From all that I can learn there is little or no private practice in Russia under the present system of government.

The retiring age for professors is 65 years. Those who have served for twenty-five years may, however, retire on a pension, which is also granted in cases of disability.

Dermatology and syphilology are taught in the fourth year, 162 hours being devoted to these subjects in addition to 15 or 20 days of practical work. Just how much didactic instruction is given I did not learn. Gonorrhea is taught as a special course by a privat-docent, 54 hours of theoretical instruction and 10 days of practical work being required. It was astonishing to learn that in the current year 53 per cent of the undergraduate medical students in Russia were women.

Graduate instruction in cutaneous and venereal diseases is given in various institutions, each student receiving 175 rubles ($87.50) per month from the state for living expenses. In a clinic in the Tarnowsky Hospital I was told that 50 graduate students were received at a time for a four month period of training. Students who are specially interested or capable may continue their graduate studies in certain institutions for from two to three years. The total number of graduate students in all branches of medicine in the Soviet Union is approximately 1,300 for the current year.

The institutions in which cutaneous and venereal diseases are treated include not only university clinics, hospitals and dispensaries, but also five large "venereological institutes" situated in Moscow, Leningrad, Odessa, Charkov and Tiflis. The institution in Moscow is known as the State Venereological Institute, and is dedicated to its founder, V. M. Bronner. The institute in Odessa honors the name of Professor Glavche in a similar manner. In line with the tremendous expansion of the five year plan, the government hopes to have twenty such institutes within the next three years.

In answer to a question regarding the total number of beds for patients with cutaneous and venereal diseases in the hospitals of the Soviet Union, I was told that there are at present 500 in the aforementioned institutes and 6,249 in various hospitals. In comparison, the number of beds devoted to this purpose in the United States is pitifully small. I was also informed that in 1929 in "Great Russia" (R. S. F. S. R.) there were 172 dispensaries for cutaneous and venereal diseases besides special departments for these diseases in general hospitals. "Great Russia" (it should be said) contains approximately half of the population of the entire Soviet Union.

Fig. 3.—Making wax casts.

Fig. 4.—Dental department.

The opportunities for clinical and laboratory research in dermatology and syphilology appear to be excellent at the present time in Russia. The clinical material in the larger cities is abundant, and the laboratory facilities are entirely adequate. The "venereological institutes" have special departments for experimental work in dermatology and syphilology and rooms devoted to serology, histopathology, mycology, bacteriology and chemistry. The large clinics are supplied with roentgen ray, ultraviolet ray and photographic apparatus, and have dental and social service departments, while the State Venereological Institute of Moscow has a work shop for making wax models of cutaneous diseases. Practically all of this I saw in my visit to Professor Podvisotskaya's clinic in the Tarnowsky Hospital in Leningrad. The entire available staff accompanied me through the institution and showed me as much attention as if I were a visiting potentate. They were kindness itself. Visits from foreign dermatologists were apparently unusual, and the oldest members of the staff could not recall that one had ever been made by an American colleague. One unusual feature caught my attention, namely, a box in which patients were requested to deposit complaints in writing regarding any feature of the treatment in the hospital which they felt could be improved.

A visit to Professor Jordan's clinic in the Second University of Moscow was also of considerable interest, although unfortunately Professor Jordan was on his vacation. The clinic was housed in a spacious building containing an adequate number of beds, apparatus and laboratories. I was interested to hear that in this clinic the roentgen rays were used to an extent approaching that in the United States. Their therapeutic employment was not merely confined to epilation in ringworm and favus and to malignant disease, but included a variety of inflammatory and other conditions of the skin. The Soviet method of spreading propaganda by posters and charts was utilized in the patients' waiting room, where plain talks were frequently given by the staff on the dangers of venereal disease.

Visits to other institutions such as the Korolienko Hospital were made, but showed nothing worthy of special mention. The clinic of Professor Meschersky in the First University was unfortunately closed for renovation. This was true also of the imposing looking Bronner Institute. Its size can perhaps be judged better by a glance at the accompanying illustration than by the statement that it contains approximately 22,000 cubic meters of space.

Information was obtained about certain diseases of interest to dermatologists, including lupus, leprosy, favus and syphilis. Lupus vulgaris was found to be relatively common in parts of Russia, as it is in certain northern countries of Europe. There are special institutions (lupusoria) for the treatment of patients with lupus vulgaris in Moscow,

Leningrad and Sverdlovsk, all of which have various types of apparatus for the employment of ultraviolet and roentgen rays. The one I visited in Moscow under the charge of Dr. Bremener was adequately equipped, but seemed small in comparison with the Finsen Institute in Copenhagen.

Leprosy was rather prevalent in view of its comparative rarity in other countries of Europe. There were ten institutions (leprosoria) scattered over the Soviet Union, having a total of approximately 1,000 patients.

Favus, which is well known to be endemic in Russia, was said to be "prevalent mostly in the frontier republics and districts and extremely frequent in some of them, especially where the cultural level of the

TABLE 1.—*Cases of Syphilis Per Ten Thousand Inhabitants in Russia*

Districts	1913	1926
Cities	180.3	120.3
Villages	53.7	56.8

TABLE 2.—*Actual Number of Cases of Syphilis in Cities*

City	1914	1925	1926	1927	1928
Moscow	27,710	22,092	16,460	11,920	10,585
Leningrad	.....	.....	11,856	9,996	3,743

population was low." For example, in 1928 a survey of 1,540 children of school age in the city of Chiva (Usbek) showed an incidence of 588 cases, or 38.1 per cent. Other parasitic diseases of the skin were also said to be relatively frequent in these frontier regions.

A few facts about syphilis in Russia may be of interest. Statistics, I was told, show a decline in the incidence of the disease in cities and a slight increase in villages from 1913 to 1926 (see tables 1 and 2). Prostitution, as the greatest source of syphilis, is combated by means of special institutions known as "prophylactoria," where the women are compelled to remain for at least a year. Here they are taught a useful occupation and are given treatment for syphilis and gonorrhea. In 1930 there were 27 of these institutions in the large cities, taking care of 2,300 women. Five of them were located in Moscow, one of which I visited. Special regulations have been enacted by the government concerning the control of prostitution and compulsory treatment of those suffering from venereal diseases. Persons contracting marriage are obliged to declare that each partner knows the state of health of the other, though no physical examinations are required by the authorities.

Patients with neurosyphilis are treated in both the clinics for neurologic diseases and those for syphilis. The "venereological institutes" have special small departments for treating this type of the disease. Neo-

Fig. 5.—Examining room in the clinic for diseases of the skin.

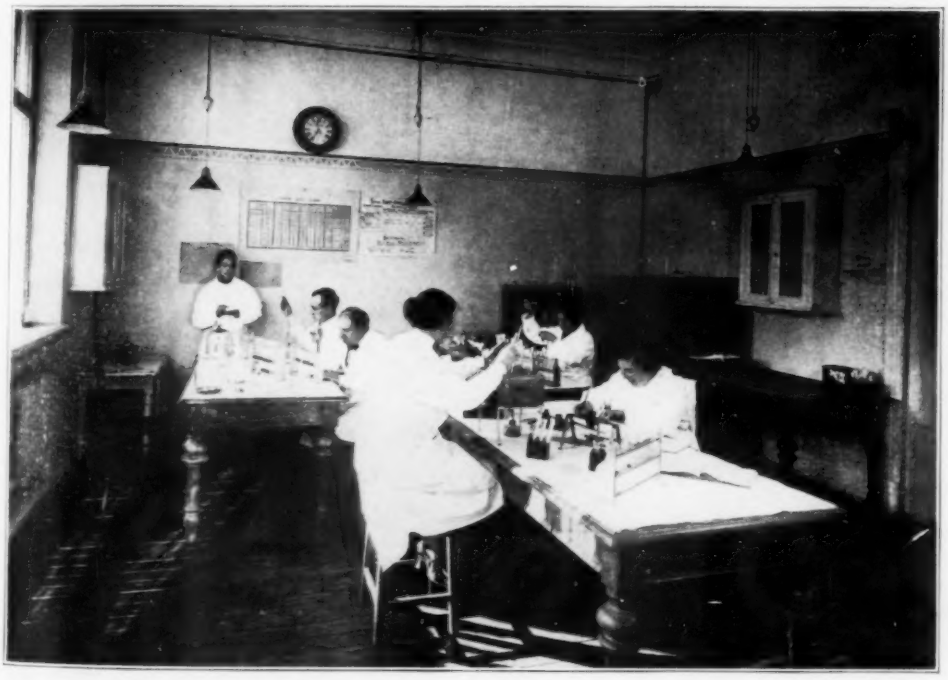

Fig. 6.—Experimental department; serological room.

arsphenamine of Russian manufacture is widely used and never in higher dosage than 6 decigrams. The use of bismuth has largely supplanted that of mercury, particularly the use of the Russian preparations known as "biochinol" and "bismutogvi." In performing the Wassermann test one or more of the precipitation tests are invariably used for comparison, including those of Sachs-Georgi, Meinicke and Kahn.

A question was asked regarding the prevalence of ulcus molle, in view of the astonishingly large number of cases (100) which I saw in the Tarnowsky Hospital. In 1929 there were 994 cases in this institution, but I was informed that this was a local epidemic, and that no other

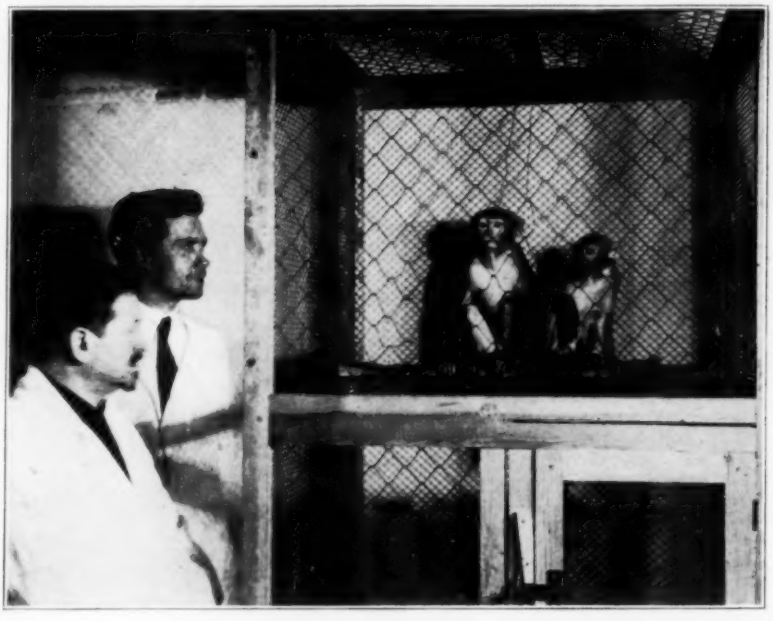

Fig. 7.—Experimental department.

cities showed any unusual prevalence of this disease. In the years from 1902 to 1913 the cities showed an incidence of from 37 to 55.8 per 10,000 inhabitants, while in 1929 this had decreased to 3.28, approximately the same as in prewar periods.

There is no national society in the Soviet Union devoted to the study of dermatology and syphilology. Local societies exist, however, in a number of the larger cities, including Moscow, Leningrad, Kiev, Odessa, Tiflis, Karkhoff, Krasnodar, Saratov and Smolensk. I was told that the society in Leningrad was founded in 1888 and the one in Moscow several years later.

There are two publications at present devoted to dermatology and syphilology. One is known as *Russky Vestnik Dermatologii* (*Russian*

Journal of Dermatology). It is published ten months each year by the Moscow Dermatological Society. It is edited by Professor Ivanov. The other journal is entitled *Venereology and Dermatology*. It is published monthly by the state and is edited by Professor Bronner. Both of these journals were founded in 1924.

It was estimated that there are about 50,000 physicians in the entire Soviet Union (U.S.S.R.) and 31,000 in Great Russia (R.S.F.S.R.). There are about 3,000 physicians working in the field of dermatology and syphilology, of whom about 570 are in Moscow and 200 in Leningrad. The number of these specialists does not appear so great in view of the fact that they all treat gonorrhea, as well as diseases of the skin and syphilis.

It might not be out of place to give a brief roster of the leaders in dermatology and syphilology in Russia, the following list being limited to those of professorial rank. Its perusal might be helpful in the study of geography, not to mention onomatology.

Moscow: A. A. Brichev, V. M. Bronner, M. P. Demianovitch, N. S. Efron, Ivanov, A. P. Jordan and G. I. Meschersky.
Leningrad: L. M. Erlich, N. G. Mgebrow, T. P. Pavlov, O. N. Podvisotskaya, A. A. Sachnovskaya and A. K. Stine.
Smolensk: Z. N. Grjebin.
Voronej: N. L. Rassiansky.
Saratov: P. S. Grigoriev.
Tomsk: A. A. Bogolepov.
Irkutsk: F. F. Udalevitch.
Omsk: A. Sarokin.
Krasnodar: V. A. Pospelov.
Rostov-on-Don: P. V. Nikolsky.
Charkov: L. A. Sabolev.
Tiflis: Burgsdorf.
Erivan: N. G. Ossipiants.
Perm: M. A. Rosentul.
Odessa: S. S. Yakovlev.
Kasan: Pilnov.
Kiev: A. Tinnenko and A. G. Lurie.
Dnepropetrovsk: Braun.
Baku: M. A. Chlenov.
Minsk: Mrngovius.
Tashkent: Milman.

One object in giving this list of representative dermatologists with the names of the cities is to enable Americans to get in touch with their Russian colleagues, if they so desire. Professor Meschersky told me that they received very little foreign literature and would welcome articles written by American dermatologists.

I wish to express my thanks to Professor Bronner for valuable data and to Drs. Michailovsky and Muskatblit for their aid in translation.

Clinical Notes

REPORT ON "A NEW FORM OF THERAPY FOR PSORIASIS"*

A. W. Stillians, M.D., and T. K. Lawless, M.D., Chicago

In the Archives for October, 1930, Campbell and Frost made a preliminary report on a new method of treatment for psoriasis, consisting of intramuscular injections of a suspension in alcohol of the patient's own scales. When obtainable, 0.2 Gm. of the scales was suspended in 20 cc. of alcohol. The dosage was from 1 to 4 cc., the average, 1.5 cc., being given every three or four days. They treated fifteen patients in this way, with varying degrees of improvement in all. The length of treatment necessary for improvement was not given, except that it varied in different cases. The signs of improvement observed were in four stages: (1) thinning and loosening of the scales; (2) paling of the centers; (3) disappearance of parts of the border, leaving only a few papules at the edge of a slightly discolored area of smooth skin, and (4) complete disappearance of these papules, leaving smooth, pigmented skin.

We have tried this method in eight cases of psoriasis, two of acute recurrence, four of the chronic guttate, discoid and circinate type and two of chronic psoriasis with disease of the joints. We did not succeed in obtaining 0.2 Gm. of scales in all cases, but we did get this much in several. We found grinding the scales difficult until we dried them first and then ground them without sand. At the convenience of our patients we gave the injections at from five to eight day intervals, in most cases once a week. The amount injected varied from 0.25 cc. to 2 cc., and the number of injections varied from three to ten, only two patients receiving less than six injections.

One of the patients with arthritis suffered severe pain in the joints with stiffness after each injection, necessitating a day in bed. No improvement could be seen after eight treatments, and the attempt was abandoned. Three patients, two with rather acute recurrences and one, a man of 37, with chronic guttate psoriasis, grew decidedly worse after the third injection, the lesions increasing in number, becoming redder and swollen and decidedly itchy. The patient with the chronic case stopped treatment at once, but the other two persisted until the sixth injection in the hope that the irritation was only preliminary to improvement; but as they continued to grow worse, they also gave up the attempt.

The other four patients in our short series made no improvement and were unwilling to continue the painful injections. The authors of this method made no mention of a preliminary stage of irritation nor of the time necessary to obtain betterment. If they had, we might have prolonged our trial of the treatment. Whether the difference in the interval between treatments could have been the cause of our failure, we do not know. We regret greatly that we must report failure in our short, perhaps too short, series.

* Submitted for publication, April 11, 1931.

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Biography

LUCIEN JACQUET *

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Lucien Jacquet was born at Saviart, France, on Oct. 29, 1860, being the oldest of eleven children. His father was originally a notary and mayor of the little community. On the advice of a friend he moved to Limoges and became a distiller. This venture was a very flourishing one, and he was able to provide good schooling for all of his offspring. Lucien was educated at the College of Saint-Martial as well as in the Medical School of Limoges. A brilliant student, he was the laureate of his class and later an intern. Late in 1883, young Jacquet moved to Paris and passed the intern examination. During his four year internship he worked under two men who influenced him to a profound degree, Besnier and Debove. The former, who united a comprehensive grasp of general pathology with a method of minute analysis of skin lesions, induced Jacquet to take up dermatology. Debove developed in Jacquet a critical spirit and also impressed on him the widespread and evil effects of alcoholism. While Besnier's intern at Hôpital Saint-Louis, Jacquet made the acquaintance of Louis Brocq, then young in dermatology but already giving promise of the remarkable career to which he was destined. Brocq and Jacquet became warm friends and remained such for life. Brocq referred to Jacquet as his oldest pupil and one of his closest friends.

In 1896 Jacquet was successful in the concours and became a Physician to the Hospitals of Paris. In 1903 he secured a service at the Saint Antoine Hospital, where he remained during the rest of his life.

Jacquet was not much concerned with case reports, with the minute description of skin disorders or with the attempt to discover new dermatologic planets. He preferred to devote his energy to the profound study of a few important problems. In this connection Brocq said: "He continually pursued the solution of interesting problems of etiology and pathogenesis, with such a degree of penetration, of observation and with such a power of methodical reasoning, that one can say without fear of exaggeration that he has changed the aspect of those disorders which he studied."

* The following articles have been written on Jaquet: Brocq, Louis: Lucien Jacquet, Ann. de dermat. et syph. **5**:569, 1914-1915; Bloch, Bruno: Lucien Jacquet, Dermat. Wchnschr. **61**:745, 1915. Siredey, Armand: Lucien Jacquet, Bull. et mém. Soc. méd. d. hôp. de Paris **44**:20, 1920.

Jacquet's first important work was done in collaboration with Sevestre. It dealt with the papular posterosive gluteal eruptions which Parrot had claimed were syphilitic. In the original paper with Sevestre, as well as in one (in 1888) of which he was the sole author, Jacquet proved the fallacy of that theory. In that same year he began to study pruritus and the pathogenesis of the pruriginous eruptions as well as the method of production of the urticarial lesions. "The true disorder is the vasomotor neurosis and the skin lesion is only secondary. The pruritus is preeruptive," he said. In 1891 and 1892 he wrote several articles on the hydrotherapeutics of lichen planus. As one of the three directors of La pratique dermatologique, he contributed three masterly chapters which are veritable monographs on prurigo, pruritus and the disorders of sensation.

His interest in pelade or alopecia areata dated from the discussion of Sabouraud's well known researches on that subject before the French Dermatological Society in 1897. Late the next year Jacquet's first paper on alopecia areata appeared. It was entitled "The Treatment of Alopecia Areata by Simple Aseptic Irritation." He wrote no less than nineteen papers on that subject. Among them were: "The Nature and Treatment of Alopacia Areata," "The Connection Between Alopecia Areata and Dental Lesions," "Disturbances in the Chemistry of the Blood and the Urine in Pelade" and "Alopecia Areata of Dental Origin." In July, 1904, Jacquet reported negative results in a large number of attempts at the inoculation of pelade, and thus did much to invalidate the parasitic theory.

His third series of researches had to do with the etiology and general pathogenesis of skin disorders and their logical treatment. In this connection he wrote about the mechanism of cutaneous hyperemia, which he christened a vasomotor pseudo-erysipelas, a cutaneous syndrome of gastro-intestinal origin which was cured by alimentary hygiene, the importance of accessory treatment in lupus, excessive gastric dilatation and overwork of tachyphagic origin, the comparative experimental influence of tachyphagia and bradyphagia, a study of professional dermatoses of the hands with reference to their etiology, pathogenesis and treatment, etc. Jacquet emphasized treatment for certain skin diseases and facial deformities by plastic massage, brushing and facial gymnastics. These measures were included in his so-called biokinetic method.

Brocq and Jacquet wrote a paper on lichen Vidal, in 1891, christening it nevrodermite chronique circonscrite. They also joined forces in writing an elementary compendium of dermatology which included five volumes. It was Jacquet who secured Brocq as the third director of La pratique dermatologique after the latter had at first refused his collaboration. Jacquet also wrote about the vernix caseosa, hidradénomes éruptifs (with Darier), bromoderma, rhinoscleroma, scleroderma, acan-

thosis nigricans and pemphigus vegetans. He first attributed Vidal's peculiar keratodermia to gonorrhea. He emphasized the importance of gonorrheal rheumatism, and described a peculiar retrocalcaneal deformity which he named talalgia and which in his opinion was due to gonorrheal infection. His contributions to the study of syphilis included his thesis, a description of the finding of Spirochaeta pallida in tertiary skin lesions (1905) and also in the suprarenals (1906). The next year Jacquet described atypical and degenerated forms of that same organism. As to the treatment for syphilis, he wrote a book with Marcel Ferrand, and in 1911 formulated an intensive plurimercurial assault for use in rebellious cases. In addition to all this he also wrote considerably along the lines of general pathology and physiology.

When convinced of the soundness of an idea Jacquet did not hesitate to defend it with the utmost vigor. In this respect Brocq considered him to be a veritable apostle. His pet ideas, among them preeruptive pruritus, the amicrobic origin of pelade, the importance of bradyphagia, the value of the biokinetic method and the antialcoholic campaign, found in him a most active and redoubtable defender. Jacquet's talent as a public speaker was due to a happy combination. Along with a pleasing voice and an animated manner he possessed an easy elocution and a well chosen vocabulary. Not a little of this success he owed to a friend who was one of the luminaries of the Comédie Française.

When convinced of the evils of alcoholism Jacquet would no longer accept financial support from his father. It is said that he experienced considerable remorse at the thought that as the son of a distiller he had benefited from what had caused so much misery to others. He resolved to devote his energy to combating this evil, and from that moment he was a real dry crusader. By his writings and by many conferences in all parts of France he endeavored to arouse his countrymen to a realization of the menace that confronted them.

Jacquet was a real friend to those permitted to know him intimately. His culture charmed those about him. A passionate admiration of art, music and literature made of him a delightful conversationalist. He often regretted not being able to devote his entire time to scientific research and intellectual pursuits.

Early in 1914 Jacquet, who had never been robust, began to fail. A serious operation afforded only temporary relief. He lingered on until Dec. 20, 1914, passing away just a few days before Fournier.

In an obituary Bruno Bloch appropriately paid tribute to Jacquet's originality and to his efforts at maintaining a close connection between dermatology and internal medicine. He ranked Jacquet among the best known members of the French school, to which opinion one cannot but hasten to subscribe.

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Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

CHANGES IN THE SKIN IN THYROTOXICOSIS, WITH A BRIEF STUDY OF THE ABSORPTION TIME OF INTRADERMALLY INJECTED SALT SOLUTION IN PATIENTS WITH THYROTOXICOSIS. J. B. YOUMANS, Am. J. M. Sc. **181**:681 (May) 1931.

In thyrotoxicosis the texture of the skin is strikingly characteristic, being smooth, silky, thin and of a close type; the skin is usually elastic, with a soft velvety feel. While this type of skin is present before the onset of the disease, these qualities become exaggerated later on during its development.

The characteristic texture is partly due to an increased blood flow through the skin, resulting from an increased metabolic rate. This increased circulation also aids in the elimination of heat and in improving the local nutrition of subcutaneous tissue.

A slight increase in pigmentation is found in these cases, the increase being particularly prominent on the exposed surfaces. It is usually diffuse, but may be of the vitiligo type. It does not occur on mucous membranes in contradistinction to Addison's disease.

While in hyperthyroid cases there may be more abundant hair on the scalp than normal, the general body hair becomes scanty, the state of hypotrichosis becoming more marked as the disease progresses, even involving the scalp.

The presence of an abnormal thyroid secretion is offered as a possible explanation for the changes in the hair in thyrotoxicosis.

Skin temperatures are distinctly elevated above normal, the increase being roughly proportional to the increase in metabolism. Coincident with a rise in temperature is an increase in moisture, giving the combination of a characteristic, warm moist skin.

The time of absorption of intradermally injected solution was greater than normal in thyrotoxicosis; this absorption rate, however, returned to normal limits following response to treatment.

ACTINOMYCOSIS STARTING AS APPENDICITIS WITH EXTENSIVE VISCERAL INVOLVEMENT: WITH A REPORT OF TWO CASES. G. M. ROBSON, Am. J. M. Sc. **181**:692 (May) 1931.

The two cases reported began with sinus formation following appendectomy. Invasion of the liver and lungs soon followed, one patient having a generalized involvement of the blood stream, with the brain, kidney and spleen affected.

The fungus was not found ante mortem in either case.

JAMIESON, Detroit.

CAROTINEMIA RESULTING FROM RESTRICTED DIET AS OBSERVED IN DERMATOLOGIC PRACTICE. OSCAR L. LEVIN and SEYMOUR H. SILVERS, J. A. M. A. **96**:2190 (June 27) 1931.

Two cases of carotinemia are reported in women who were not diabetic. In one, the pigmentation appeared eight months after a restricted diet was instituted, and in the other, the pigment appeared in ten months' time. In both patients the involvement was of the exposed portions of the body, and the pigmentation cleared up after a few weeks of proper dietary management. The literature on carotinemia is adequately reviewed, and the differential diagnosis from icterus is considered. The endogenous pigment carotene is found in common foods, such as carrots, pumpkins, yellow turnips and squash, parsnips, spinach, lettuce, oranges and eggs.

Reappearance of Chickenpox Lesions Following Administration of Diphtheria Toxin-Antitox. William M. Donald and William L. Brosius, J. A. M. A. **96**:2196 (June 27) 1931.

A 5 year old boy was seen on Dec. 15, 1930, with an eruption of chickenpox which ran a normal course. One month later, he received an immunizing dose of diphtheria toxin-antitoxin in the gluteal region. One week later, he received the second injection, and the following day there developed lesions of chickenpox on the lower part of the back, hips and posterior surfaces of both thighs. These healed in two weeks.

The Problem of Arsphenamine-Resistant Syphilis. Report of a Case. Thomas H. Miller, J. A. M. A. **97**:11 (July 4) 1931.

This is a report of a case of primary syphilis which was resistant to arsphenamine and which healed after sulpharsphenamine and bismuth were given intramuscularly. The arsphenamine, when tested, proved to be sufficiently active trypanocidally. The literature on arsphenamine-resistant syphilis is critically reviewed, and the author concludes from his study that the individual is the responsible etiologic factor in resistance, although the mechanism is not known. Arsphenamine-resistant syphilis may manifest itself in the persistence of syphilitic lesions; in a persistently positive Wassermann reaction of the blood, or in the persistence of Spirochaeta pallida in the lesions after so-called adequate treatment. Apparently, there is an increase of the condition in France and Germany, but not in the United States.

Comparative Studies on Calcium Gluconate and Other Calcium Salts. Arnold L. Lieberman, J. A. M. A. **97**:15 (July 4) 1931.

Experimental studies indicate that calcium gluconate can be given subcutaneously and in adequate amounts without causing irritation or necrosis. Intravenous therapy is dangerous because of the menace of sudden intravascular clotting. From 3 to 4 Gm. a day after meals is the most favorable dose. When given on an empty stomach, it is apt to produce diarrhea. The blood calcium attains its maximum elevation within one hour after injection, and four hours after the oral route. Lactate salts showed relatively evanescent effects as compared to the gluconates. Digitalis effects were noticed with both, more slowly with the gluconates, but when once produced by the gluconates, they persisted for a longer time.

Studies in Thrombo-Angiitis Obliterans (Buerger): VIII. Effect of Thyroid Administration on Blood Volume. Samuel Silbert and Mae Friedlander, J. A. M. A. **97**:17 (July 4) 1931.

The authors have previously shown that patients with thrombo-angiitis obliterans have a low blood volume and a low basal metabolism. They treated fourteen men with typical cases, ranging in age from 25 to 40 years, for periods of from four to eight months, and determinations were made at monthly intervals of the basal metabolism, blood chemistry and clinical course. After the administration of thyroid the low blood volume increased to normal, but fell slowly as the drug was continued. On withdrawal of the drug, the blood volume fell to its original low figure. When the blood volume was increased, the total proteins, calcium and cholesterol figures diminished to normal and increased when thyroid was withdrawn.

The Germicidal and Therapeutic Applications of Soaps. John E. Walker, J. A. M. A. **97**:19 (July 4) 1931.

Walker concludes from his studies that soaps are strongly bactericidal against pneumococci, streptococci, meningococci, gonococci, diphtheria bacilli, influenza

bacilli and Spirochaeta pallida. Unfortunately, soaps do not destroy staphylococci or typhoid bacilli. The germicidal activity of soaps compares favorably with that of many of the popularly used synthesized chemicals. A 2 per cent solution of sodium oleate is beneficial as an irrigation or as wet dressings for ulcerated surfaces. The concentration of soap in an ordinary thick lather used in washing the hands is about 1 : 12, and concentrations of soap of from 1 : 80 to 1 : 1,280 destroy bacteria.

CHROME POISONING WITH MANIFESTATIONS OF SENSITIZATION. ADELAIDE ROSS SMITH, J. A. M. A. **97**:95 (July 11) 1931.

The patient whom this report concerns gave a history of previous attacks of hay fever and asthma. After exposure to a chrome compound, a dermatitis developed on the hands without other symptoms, followed by an interval of freedom from exposure during which the dermatitis cleared. One month later, after a second exposure to chrome, there developed chrome holes, with systemic symptoms of fever, asthma, myositis and nephritis with glycosuria. After recovery, an intradermal injection of a chrome solution produced a focal reaction on the hands, a lighting up of the patch test site and erythema with urticarial wheals on the buttocks, evidences of sensitization to chrome.

A CASE OF POLYNEURITIS FROM THALLIUM ACETATE. CHARLES L. SHORT, J. A. M. A. **97**:101 (July 11) 1931.

Another case of poisoning from "Koremlu Cream" is reported. The patient, a woman, complained of a polyneuritis; the diagnosis was made after the hairs of the scalp began to fall. The symptoms began after she had used the cream daily for one month. She was treated with calcium lactate, and improved slowly. In this patient there is a possibility of a permanent damage to the extensors and flexors of the feet.

THE INCIDENCE OF EMBOLISM IN TREATMENT OF VARICOSE VEINS WITH SCLEROSING SOLUTIONS: REPORT OF A FATALITY. I. SIVERMAN, J. A. M. A. **97**:177 (July 18) 1931.

Varicosities in a woman, aged 44, were treated with a total of 25 cc. of 25 per cent sodium chloride solution in five different places. Two days later she complained of severe pain about the areas of injection, with dusky discoloration, hardness and tenderness. After eight days in bed she sat up, and died immediately of a pulmonary embolus. A study of the literature has revealed a total of nineteen cases of embolus following the injection treatment, of which fourteen were fatal. The author thinks that from 5 to 10 cc. of the drug at a single point is the proper dose.

AN UNUSUAL SKIN REACTION TO EPINEPHRINE. R. W. LAMSON and S. O. CHAMBERS, J. A. M. A. **97**:314 (Aug. 1) 1931.

A man, aged 31, who suffered from asthma, had been giving himself injections of a 1 : 1,000 solution of epinephrine hydrochloride. At the sites of injection, there appeared a wheal-like, superficial nodule of an indolent red color. This changed to a superficially crusted ulceration, which healed with scar formation, some of which was smooth and level; other parts were depressed, and some were hypertrophic. The clinical picture had features of, and had to be differentiated from, acne varioliformis, dermatitis factitia and dermatitis herpetiformis. A photograph illustrating the scars was not very clear.

RATTNER, Chicago.

HYPERSENSITIVENESS TO SOLUBLE SPECIFIC SUBSTANCES FROM YEAST-LIKE FUNGI: I. ANAPHYLAXIS; II. EYE HYPERSENSITIVITY. H. D. KESTEN and E. MOTT, J. Exper. Med. **53**:803 and 815 (June) 1931.

Passively sensitized guinea-pigs developed a rapidly fatal anaphylactic shock following the injection of polysaccharide fractions from each of five yeastlike fungi.

In guinea-pigs actively sensitized with killed Monilia psilosis, the polysaccharide fraction caused death. Attempts to sensitize actively with this fraction were unsuccessful.

Heat-killed Monilia psilosis was injected into the anterior eye chamber of twelve rabbits. Intravenous injection of the polysaccharide fraction from this organism produced a reaction in the sensitized eye of five of the twelve rabbits.

YAWS INCIDENCE IN THE SOUTH PACIFIC. S. M. LAMBERT, J. Trop. Med. **34**:117 (May 1) 1931.

Among the natives of the South Pacific Islands yaws was found to be almost universally distributed. On the other hand, syphilis practically does not exist, and the writer thinks that yaws might possibly immunize against syphilis in the natives of this region.

The disease is disappearing since the advent of arsphenamine, although mass treatment in active cases did not eradicate the disease.

The suggestion is made for mass treatment of all youths up to the age of 17 for the purpose of total eradication of yaws.

JAMIESON, Detroit.

PARAPSORIASIS ALMOST CURED BY GOLD THERAPY. GOUGEROT and CARTIAUD, Bull. Soc. franç. de dermat. et syph. **38**:606 (April) 1931.

The patient had a clinically typical eruption of this disease, with a negative Wassermann reaction and a strongly positive tuberculin test. Therapy with a gold compound was given from November, 1929, to April, 1930, with decided improvement.

ANGIOKERATOMA (MIBELLI) ASSOCIATED WITH NODULAR AND PAPULONECROTIC TUBERCULIDS. GOUGEROT, P. BLUM and BRALEZ, Bull. Soc. franç. de dermat. et syph. **38**:610 (April) 1931.

The authors presented a patient with angiokeratoma of the soles and tuberculids on the lower extremities. The Mantoux test was strongly positive. The case was presented as further evidence of the tuberculous origin of angiokeratoma.

TWO CASES OF "PHOTO-MICROBIC ECZEMA" OF THE FACE. JAUSION, GERVAIS and LARTIGUE, Bull. Soc. franç. de dermat. et syph. **38**:616 (April) 1931.

Two cases of a squamous type of eczema of the face are described in which the disease appeared after a streptococcic impetigo at that locality. Both patients were exposed constantly to sunlight. Intradermal injections of streptococcic antigen gave strongly positive responses; tests for other microbes resulted negatively. The authors think that insulation was a predisposing factor in these cases. Properly speaking, they were not cases of actinodermatitis. The conception of the authors as regards photomicrobic dermatitis is that after long exposure to various electromagnetic modalities (sunlight, heat, roentgen rays) the skin becomes sensitive to various antigens (microbic and chemical antigens, the products of digestion, etc.).

STAPHYLOCOCCIC ACRODERMATITIS AND RAYNAUD'S DISEASE. JAUSION, LARTIGUE and GERVAIS, Bull. Soc. franç. de dermat. et syph. **38**:624 (April) 1931.

The authors' patient presented recurrent erythematosquamous eczema of the hands with various forms of pyoderma (paronychia, Bockhardt's impetigo, etc.)

of the same regions. From the suppurative lesions Staphylococcus aureus was recovered. In addition, the patient had attacks of angiospasm of the Raynaud type. There appeared to be a connection between the vascular disturbance and the other manifestations.

POIKILODERMA EN PLAQUE. SÉZARY, GALLERAND and LÉVY-COBLENTZ, Bull. Soc. franç. de dermat. et syph. **38**:632 (April) 1931.

The patient, a woman, aged 42, presented a vast unilateral thoracic placard exhibiting a dry, yellowish-red, desquamating, finely plicated surface. There were also irregularly distributed areas of telangiectasia and atrophy. There were no subjective symptoms. Microscopic examination showed the characteristic architecture of poikiloderma. No similar case of poikiloderma seems to have been reported.

CHRONIC STREPTOCOCCIC ERYTHRODERMA. SÉZARY and HOROWITZ, Bull. Soc. franç. de dermat. et syph. **38**:644 (April) 1931.

Sézary and Horowitz describe an exfoliative dermatitis of four years' duration, which disappeared promptly following the use of yellow mercuric oxide ointment. The etiologic significance of streptococci was suspected because of retro-auricular and umbilical lesions. Streptococci were cultivated from the skin by the pipet method.

ARSPHENAMINE DERMATITIS TREATED BY SPLENIC EXTRACT. G. HUFSCHMITT, Bull. Soc. franç. de dermat. et syph. **38**:702 (April) 1931.

Hufschmitt reported a severe case of arsenical erythroderma in which treatment with splenic extract, begun about a week after the onset, led to rapid cure. The extract was given subcutaneously in progressively increasing daily doses. An attempt was then made to protect the patient against arsphenamine by giving simultaneous treatment with splenic extract; however, this was a failure as signs of arsenic intolerance recurred.

SQUAMOUS CELL EPITHELIOMA DEVELOPING ON ULCERATED BUCCAL LICHEN PLANUS. B. SAAD, Bull. Soc. franç. de dermat. et syph. **38**:705 (April) 1931.

The author reported a case of buccal lichen planus of three years' duration in which there was an ulceration on the right cheek with the development of cancer. This diagnosis was confirmed by biopsy. On the left cheek a characteristic arborescent patch of lichen planus was still present.

RECURRENCE OF SYPHILIS FOLLOWING TREATMENT WITH NEOARSPHENAMINE. LACASSAGNE and FRIESS, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:476 (April) 1931.

The authors' patient was given a total dosage of 5.70 Gm. of neoarsphenamine in the treatment for a penile chancre. Fifteen days after the cessation of treatment, a disseminated erythematosquamous eruption with mucous patches developed. The eruption receded under bismuth therapy.

THE DIFFERENTIAL DIAGNOSTIC VALUE OF FREI'S REACTION AND THE THERAPEUTIC TEST WITH DMELCOS' VACCINE IN NICOLAS-FAVRE'S DISEASE AND CHANCROIDAL BUBO. J. GATÉ and J. CHARPY, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:487 (April) 1931.

A propos of two cases of inguinal adenitis seen by the authors in which the differential diagnosis was delicate, a résumé of the value of biologic tests and other measures in the differentiation of the aforementioned diseases was discussed.

In the first place, the clinical features are of decided significance, especially whether the adenitis is monoglandular or polyglandular, and whether or not a concomitant iliac adenopathy is present. The two latter features indicate lymphogranuloma inguinalis. Among the laboratory procedures, the finding of Ducrey's bacillus (with the rare exception of mixed infections) is decisive. The intradermal reaction of Dmelcos has considerable value. The result of inoculation of pus from the bubo is decisive only if a chancroidal ulcer develops. Frei's reaction is theoretically specific; practically, however, it is not always so. Doubtful reactions are sometimes obtained and give rise to considerable difficulties in interpretation. Finally, the therapeutic test by intravenous injections of Dmelcos' vaccine gives positive proof of a Ducrey infection if the lesions disappear.

ERYTHEMA MULTIFORME ASSOCIATED WITH NICOLAS-FAVRE'S DISEASE. NICOLAS, M. FAVRE and F. LEBEUF, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:509 (April) 1931.

During the past two years the authors have seen fourteen cases of this disease, two of which showed erythema multiforme and one, erythema nodosum. This fact indicates a systemic involvement in Nicolas-Favre's disease, a point already evidenced by specific biologic reactions.

THE PRESENCE OF GOLD IN THE SPINAL FLUIDS IN PERSONS TREATED BY INTRAVENOUS INJECTIONS OF GOLD. F. LEBEUF, H. MOLLARD and P. PAUGET, Bull. Soc. franç. de dermat. et syph. (Réunion dermat. Lyon) **38**:513 (April) 1931.

The authors found gold in the spinal fluid in three patients who had received injections of that metal. However, the spinal fluid contained gold only after a considerable quantity had been injected, and the amount in the fluid was proportional to the total dose received.

A SENSITIZATION DERMATITIS, OCCURRING IN A PATIENT WITH LICHEN PLANUS AFTER THE SECOND DOSE OF ACETYLARSAN. BIOLOGIC TESTS. J. GATÉ, H. THIERS, J. CHARPY and P. CUILLERET, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:516 (April) 1931.

The patient presented a severe exfoliative dermatitis. While the disorder was active, the authors obtained a passive sensitivity to acetylarsan in a guinea-pig. This was successful with serum from bullae and the patient's blood. After the disappearance of the cutaneous lesions there were positive reactions to scratch and intradermal tests with acetylarsan.

ERUPTION DUE TO SODIUM SALICYLATE. BIOLOGIC TESTS SHOWING ITS TOXIC NATURE. J. GATÉ, M. BERNHEIM, H. THIERS and P. GUILLERET, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Lyon) **38**:518 (April) 1931.

In the authors' patient a severe erythematous and bullous eruption developed from intensive treatment with sodium salicylate. The authors were unable to sensitize guinea-pigs passively with the patient's serum, and concluded that the cutaneous manifestations were of a toxic, rather than an allergic, nature.

PURULENT INGUINAL ADENOPATHIES. L. M. PAUTRIER, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:524 (April) 1931.

In opening the dermatologic conference of March 8, 1931, at Strasbourg, consecrated to lymphogranulomatosis inguinale, chancroid and ulcus vulvae acutum, Pautrier spoke chiefly of the former condition and recited the history of the recognition of that disorder by Nicolas and Favre whose first work on it was

published in 1913. Since then there has been general acceptance of their observations. Pautrier, however, remarked that the name lymphogranuloma inguinale is unsatisfactory, and that the most desirable term for the present, at least, is Nicolas-Favre's disease.

A Variety of Nicolas-Favre's Disease with the Formation of Abscesses. L. M. Pautrier, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:531 (April) 1931.

Pautrier discussed a case seen in his service in which large purulent collections developed. The simulation of chancroidal bubo caused diagnostic difficulty, which was cleared up by laboratory examination and allergic tests. In this instance the disease responded to roentgen therapy.

Anorectal Stricture: An Atypical Manifestation of Nicolas-Favre's Disease. J. Nicolas, M. Favre, G. Massia and F. Lebeuf, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:533 (April) 1931.

The authors found Frei's reaction positive in two cases of anorectal stricture. In one of the cases the histologic condition was that of Nicolas-Favre's disease. Other authors have reported similar observations, and this collected experience leads to the conclusion that some cases of rectal stricture are part of the syndrome of lymphogranuloma inguinale.

Four New Cases of Anorectal Syphiloma and Esthiomene with Positive Frei Test. O. Jersild, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:537 (April) 1931.

Jersild has personally tested seven patients with these conditions with Frei's antigen, and all gave positive reactions. He collected sixteen cases from the literature with the same finding. From this he concludes that esthiomene, anorectal syphiloma and Nicolas-Favre's disease have the same cause.

Nicolas-Favre's Disease. Gans, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:544 (April) 1931.

Gans gave a short description of the histology of an initial lesion and of an erythema nodosum efflorescence occurring in the same patient. In the former condition he noted acanthosis and a pronounced infiltration of the upper and middle cutis. Because of its bandlike topography, this infiltration recalled that of lichen planus, but it was composed chiefly of polymorphonuclear leukocytes. There was considerable edema of the upper cutis and epidermis. In the nodose lesion the chief feature was a perivascular polymorphonuclear infiltration.

The Intradermal Reactions in Nicolas-Favre's Disease. J. Nicolas, M. Favre and F. Lebeuf, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:552 (April) 1931.

The authors discuss the diagnostic reactions in this disease and conclude that the intradermal reactions employed (with the various antigens so far suggested) have great diagnostic value. The reaction is en rapport with the involvement of the skin overlying the affected glands and the systemic symptoms. Radiotherapy intensifies the reaction. Specific allergy is demonstrable a variable time after the initial infection and persists indefinitely. Causes of error residing in the technic of preparation of the antigen are to be avoided.

CUTANEOUS REACTIONS TO NONSPECIFIC ANTIGENS (CHANCROIDAL AND TUBERCULIN) IN NICOLAS-FAVRE'S DISEASE. J. NICOLAS, M. FAVRE and F. LEBEUF, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:558 (April) 1931.

In all cases examined by the authors, the Pirquet test was positive. All patients gave negative responses to chancroidal antigen.

EXPERIENCES WITH THE SPECIFIC INTRADERMAL TEST IN NICOLAS-FAVRE'S DISEASE. HELLERSTRÖM, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:560 (April) 1931.

Hellerström noted focal reactions in some cases. He also admits the possibility of a specific anergy in this disease, but for the present this must be accepted with reserve. In his experience, Frei's test has proved absolutely specific.

RAVAUT'S INTRADERMAL TEST FOR CHANCROID. O. JERSILD, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:563 (April) 1931.

Ravaut obtained his antigen by scraping the chancroid, suspending the material in saline solution and injecting it intradermally. A positive reaction shows an ulcer from which Ducrey's bacillus can be obtained; a doubtful reaction presents a papule without necrosis, and a negative reaction shows no alteration at the site of injection. Jersild corroborates Ravaut's observations; in the present article, however, he especially discusses the doubtful reactions, which occur in about 50 per cent of the cases. For him, these doubtful reactions indicate an allergic state but not a specific allergy. It is an allergy to a nonspecific antigen present in all bubos; hence the doubtful reaction has no diagnostic significance.

INTRADERMAL REACTIONS TO VAGINAL SCRAPINGS. L. M. PAUTRIER, R. GLASSER and P. LANZENBERG, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:568 (April) 1931.

Ravaut recently described an intradermal reaction to vaginal scrapings in two women who apparently were carriers of the Ducrey bacillus. Pautrier and his collaborators carried this work further by investigating the reaction in twenty-four prostitutes who had no genital lesion. In eight of the tested persons these investigators obtained a positive reaction, but these reactions were either papular or erythematous, never chancroidal. The authors believe that it is necessary to interpret such reactions with reserve. They are of dubious significance. Only a frankly ulcerative reaction with recovery of Ducrey's bacillus can be accepted unreservedly as positive.

A VIRUS, PATHOGENIC FOR MONKEYS, IN CERTAIN VENEREAL BUBOS. LEVADITI, RAVAUT LÉPINE and SCHOEN, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:576 (April) 1931.

The authors report the results of intracerebral inoculations in monkeys of material from lymphogranuloma inguinale bubos. Their conclusions are that these infected glands contain a filtrable virus that is pathogenic for some species of monkeys. The virus is destroyed by glycerin but remains active in congealed cerebral tissue. When it is injected intracerebrally into monkeys, it causes either a fatal meningo-encephalitis or a clinically inapparent but highly virulent disease of the central nervous system.

RADIOTHERAPY OF SUBACUTE LYMPHOGRANULOMA INGUINALE. P. FERNET, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **38**:585 (April) 1931.

Roentgen therapy is most beneficial in the early nonsuppurative stages of the disease. It has been used at the St. Louis Hospital, Paris, in twelve cases, with

cures in eight, marked improvement in three and slight benefit in one. A filtered ray (0.5 zinc) was employed. A second dose, administered six or eight weeks after the first, was necessary in some cases. In the discussion, Nicolas said that his results with radiotherapy in this disease were very satifactory. Hellerström mentioned therapeutic success with intravenous injections of the specific antigen obtained from the bubos.

MICHAEL, Houston, Texas.

ACRODYNIA IN INFANTS. J. GIRARD, Progrès méd., Feb. 28, 1931, p. 389.

The author describes a case of acrodynia in an infant 3 months old. The cutaneous symptoms followed convulsions and cerebral symptoms analogous to encephalitis. On the basis of this case and others taken from the literature, he believes that acrodynia is only a cutaneous symptom of encephalitis.

SANFORD, Chicago [AM. J. DIS. CHILD.].

SODIUM CHLORIDE. OTTO KESTNER, Dermat. Wchnschr. **92**:641 (May 2) 1931.

The author describes the importance of sodium chloride in the human organism from the standpoint of a physiologist. He points out that it is the only mineral that must be added to the diet, and that it is especially necessary when considerable perspiration occurs. He does not deny the possibility of a good therapeutic effect from a salt free diet in special instances, but the general use of such a diet by the laity is considered dangerous.

A CASE OF VACCINE INFECTION IN A PATIENT WITH ECZEMA. HEINZ SPRENGEL, Dermat. Wchnschr. **92**:645 (May 2) 1931.

A woman, 42 years of age, had been successfully vaccinated on two previous occasions. Following the vaccination of her youngest child vaccinia developed limited to areas affected by eczema, the lesions being especially numerous on the flexor surface of the left arm, with which she usually carried the baby. The course was mild and afebrile.

THE PATHOLOGY OF THALLIUM ACETATE POISONING. J. v. VÁSÁRHELYI, Dermat. Wchnschr. **92**:649 (May 2) 1931.

Using white rats as subjects, the author followed the course of chronic thallium acetate poisoning and found in the results of biopsies evidence of atrophic and inflammatory changes in the skin. He considers the epilation to be due to the local effect of the drug rather than to an indirect effect produced by changes in the glands of internal secretion.

SEVERE ANGIONEUROTIC SEIZURE DUE TO ACETARSONE. FRANZ v. VERESS, Dermat. Wchnschr. **92**:653 (May 2) 1931.

A 32 year old man suffering from lupus erythematosus had previously been given two series of neoarsphenamine treatments. Following the last injection, six months before, he had had a severe nitritoid reaction. He was given an acetarsone tablet (0.25 Gm.) once a day, and several hours following the third or fourth dose, a nitritoid reaction developed exactly similar to the previous one.

BISMUTH AS A PROPHYLACTIC AGENT. E. SONNENBERG, Dermat. Wchnschr. **92**: 655 (May 2) 1931.

Weekly injections of a bismuth preparation were given to a group of prostitutes. No chancres developed during the injections after the fifth week. A primary infection occurred in two of them, in the third and fourth week, respectively, after cessation of treatment.

ZOSTER GENERALISATUS (VARICELLOSUS), LEUKEMIA LYMPHATICA AND ARSENIC. ARIE DOSTROWSKY, Dermat. Wchnschr. **92**:685 (May 9) 1931.

A man, 55 years of age, who had suffered from lymphatic leukemia for two years had received considerable radiotherapy, as well as arsenic administered by mouth and by injection. Severe hemorrhagic and necrotic zoster developed with a generalized varicelliform eruption. A son who had been isolated at home with his father following scarlet fever developed a typical varicella twenty-five days after the appearance of zoster in the father.

A second patient, a 62 year old man, is described, who also had lymphatic leukemia. This patient also had received radiotherapy and considerable arsenic by mouth. A papular and bullous eruption developed on the hands and ankles, which disappeared in the course of further irradiation of the spleen, lymph glands and bones.

A CASE OF ANONYCHIA TOTALIS CONGENITA. A. LISTENGARTEN, Dermat. Wchnschr. **92**:691 (May 9) 1931.

A patient is described in whom the nails of the fingers and toes were absent from birth. The nail-beds remained, and roentgenograms showed no bone changes. There were no other instances of the condition in the family.

HEMATOLYMPHANGIOMA OF THE TONGUE. ILSE FRIEDHEIM, Dermat. Wchnschr. **92**:717 (May 16) 1931.

The author describes a patient with a cystic lymphangioma of the tongue that clinically suggested the association of hemangioma. Biopsy showed a lymphangioma in which some of the cysts were filled with blood owing to breaking of the wall of a capillary blood vessel. There was no evidence of hemangioma microscopically.

FUNCTIONAL TESTS TO DETERMINE THE DISPOSITION TO THE DEVELOPMENT OF ECZEMA. KARL STEINER, Dermat. Wchnschr. **92**:723 (May 16) 1931.

The author criticizes the paper of Frei, Mayer and Halle, entitled, "Concentration Tables for Eczema Tests," in which they attempted to determine the strength of thirty-three inorganic substances that irritated apparently normal skin. He especially objects to the fact that tests on an insufficient number of subjects were made (five males and five females). The patch tests are only of value in congenital hypersensitiveness. In an added note the more recent paper by Stauffer, "The Eczema Tests," is also objected to on the ground that the tests are of value from a theoretical rather than a practical standpoint.

THE DISTRIBUTION OF LYMPHOGRANULOMA INGUINALE. F. BERNSTEIN, Dermat. Wchnschr. **92**:731 (May 16) 1931.

The first cases of lymphogranuloma inguinale reported in Germany were all from seaport cities. During the past two years, three patients with typical cases were treated in the Düsseldorf clinic. None of these cases presented complications such as exanthems or erythema nodosum, and the primary lesion was not observed in any of them. It appears that the disease is spreading.

GUMMAS OF THE SKIN AFTER MALARIA THERAPY. K. SCHREINER, Dermat. Wchnschr. **92**:764 (May 23) 1931.

Though cutaneous lesions do occur in patients with cerebrospinal syphilis, they are unusual. In a 28 year old man a gummatous eruption on the scalp developed three years after malaria treatment. The eruption was apparently started by a pyogenic folliculitis. The literature concerning the development of late syphilis of the skin following malaria therapy is reviewed. The author believes that this form

of treatment may either directly or indirectly predispose the patient to the development of tertiary skin lesions.

THE GRANULAR FORM OF SPIROCHAETA PALLIDA. NICOLAS Z. JOANNIDÈS, Dermat. Wchnschr. **92**:775 (May 23) 1931.

The author points out that he described in 1906 a granular form of Spirochaeta pallida which he called sigmata. These "S" forms were found, along with the ordinary forms, in the early lesions in patients undergoing treatment with mercury, and they were also found in late syphilitic lesions.

THE LOCAL TREATMENT OF FURUNCULOSIS WITH PURE SULPHONATED BITUMEN, N. F. L. N. MASCHKILLEISSON, Dermat. Wchnschr. **92**:776 (May 23) 1931.

The treatment of furuncles by painting the lesions daily with pure sulphonated bitumen, N. F., is considered the most satisfactory available. It decreases the likelihood of new lesions, heals the lesions more rapidly and is painless and inexpensive.

THE EARLY DEMONSTRATION OF SPIROCHETES IN SCROTAL LESIONS OF RABBITS INOCULATED WITH MATERIAL FROM THE POPLITEAL GLANDS OF RABBITS WITH LATENT SYPHILIS. RUDOLF STREMPEL, Dermat. Ztschr. **61**:1 (May) 1931.

Levaditi and his associates inoculated the scrotums of normal rabbits with material from the popliteal glands of rabbits with latent syphilis. They first observed typical spirochetes in the lesions forty-five days after inoculation. The organisms were seen associated with unusual forms, which they considered developing stages. The author repeated this work, and was able to demonstrate normal spirochetes by silver impregnation of sections from the vicinity of the inoculation twelve days after infection. These normal spirochetes were few, and were not associated with the aberrant forms which he first observed thirty-five days after infection. He concludes that these aberrant forms are due to degeneration of the normal organisms rather than a part of a development cycle.

THE MEINICKE CLEARING REACTION IN COMPARISON WITH THE ACTIVE COMPLEMENT-FIXATION OF BRUCK AND THE ORIGINAL WASSERMANN REACTION. C. BRUCK and K. BEHRMANN, Dermat. Ztschr. **61**:7 (May) 1931.

The serums of 929 patients were the basis for this report. There were variations in 143 instances. The Meinicke reaction gave 93 more positive tests than the Wassermann and 54 more than the active complement-fixation test in undoubted cases of syphilis. It also showed about twice as many nonspecific reactions as either of the others: 0.8 per cent, compared with 0.5 per cent for the Wassermann and 0.4 per cent for the Bruck test.

A NONSYPHILITIC CONDITION OF THE GENITALIA RESEMBLING SECONDARY SYPHILITIC PAPULES. EDMUND HOFMANN, Dermat. Ztschr. **61**:10 (May) 1931.

Four patients are described in whom an inflammatory condition of the labia developed, with eroded and warty papules and superficial ulcerations. The appearance suggested secondary syphilis, which was ruled out by further study. Sections of material removed in one of the cases had an appearance similar to that of the pseudosyphilis described by Lipschütz, showing bizarre acanthosis with slight invasion of the epidermis by leukocytes and an increase of the blood vessels in the corium, which showed mesarteritis and a moderate infiltration including many plasma cells. In all of the patients the condition cleared up rather rapidly

under indifferent therapy, boric acid solution being used principally. The condition is considered to be due to lack of proper hygiene.

EXPERIMENTS WITH THE SO-CALLED ANTICUTIN AND PROCUTIN IN THE SERUM OF PATIENTS WITH SARCOID. A. KISSMEYER and GERDA SCHULTZ, Dermat. Ztschr. **61**:18 (May) 1931.

Serum obtained from nine patients with sarcoid was mixed in various dilutions with old tuberculin. This mixture was injected into patients with positive Pirquet reactions to determine whether the severity of the reaction was influenced. The reaction was decreased by the serum from four of the patients, all of whom showed the "lupus pernio" type of Boeck sarcoid, demonstrating the presence of anticutin. The presence of anticutin, however, is not to be considered positive evidence of a tuberculous etiology of sarcoid.

EXPERIMENTAL RESEARCH CONCERNING SYPHILITIC LYMPH GLAND INFECTION OF APES. PANAGIOTIS B. PHOTINOS, Dermat. Ztschr. **61**:27 (May) 1931.

A male monkey (Cercocebus) was inoculated scrotally with material from a rabbit's chancre and from the secondary genital papules of a patient. Twenty-two hours later, the inguinal glands were excised and injected subscrotally into a rabbit. Eight weeks later, typical chancres appeared, proved by demonstrating the spirochetes both in dark-field examination and by silver impregnation of sections. The same procedure was followed in a second monkey, except that the glands were excised five hours after inoculation. The transfer to the rabbit's scrotum also resulted in the development of a typical chancre.

GRANULOMA TELANGIECTODES. PAUL BALOG, Dermat. Ztschr. **61**:31 (May) 1931.

The author studied five cases of granuloma telangiectodes histologically and concludes that the process is primarily a proliferation of endothelial cells. The banal inflammatory reaction often seen microscopically is to be interpreted as a secondary occurrence. The cause of the proliferation is probably an inflammatory irritation.

L. TAUSSIG, San Francisco.

XANTHELASMA TREATED WITH THORIUM X: CASE REPORTS. W. MEERHOFF, Klin. Monatsbl. f. Augenh. **85**:661 (Nov. 28) 1930.

Operative removal of xanthelasmas does not prevent recurrences, nor is it practical in cases with multiple lesions. As the use of roentgen rays and radium is too risky for the eye, Loehlein experimented with ray treatments of thorium X and with an ointment and also an aqueous solution of thorium X. The solution must be freshly prepared in each instance, because the activity of the alpha rays is at its height twenty hours after preparation, sinks rapidly afterward and is exhausted after two weeks.

Meerhoff reports six cases in which the patients were treated at Loehlein's clinic by placing small pieces of one layer of gauze, the size of the xanthelasmas, over these formations and by moistening them with the solution from four to five times. The treatment was continued until a cutaneous reaction appeared. The doses administered could be figured out, since the quantity of "es-E" (Emanations Einheiten or emanation units) contained in each cubic centimeter of the solution was known. Most of the xanthelasmas disappeared completely, while some of them remained visible in the form of faint yellow spots. The treatment with thorium in any form, including the aqueous solution, will not prevent recurrences in some cases. It is recommended, however, on account of its simplicity, painlessness and lack of risk. Large doses have not been used up to this time.

K. L. STOLL [ARCH. OPHTH.].

MALIGNANT NEOPLASMS CAUSED BY ULTRAVIOLET IRRADIATION OF WHITE MICE. C. W. HERLITZ, I. JUNDELL and F. WAHLGREN, Acta pædiat. **10**:321 (March 15) 1931.

Three groups of animals were studied, two with various dosages of light from a mercury vapor quartz lamp, and the other as a control. Some of the irradiated animals died of intercurrent infection or other accidental causes after ten or twenty weeks of treatment. These showed early changes in the skin of the exposed parts poorly protected by body hair: hyperkeratosis, epithelial hyperplasia and sometimes ulceration. As the duration of irradiation increased to around thirty weeks, there invariably developed epidermal neoplasms, usually carcinomatous but sometimes more closely resembling some form of sarcoma. Instances of metastases to lymph nodes and the lungs were observed. The control series showed no tumor formation.

McINTOSH, New York [AM. J. DIS. CHILD.].

DERMATOLOGIC ABSTRACTS

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

RADIATION THERAPY IN TREATMENT OF CANCER OF MOUTH AND LIPS. J. M. MARTIN, Radiology **16**:881 (June) 1931.

Martin believes that cancer of the mouth and of the lower lip in the early stages does not frequently metastasize. For clinical convenience, all cases of cancer of the lower lip are roughly classified under three stages. In the first stage are grouped all cases in which the lesions are confined to the superficial tissues; that is, to the mucocutaneous structures proper. The second stage includes all lesions that have become more extensive, with the induration and ulceration more pronounced and the subcutaneous structures involved without evidence of invasion of the lymph nodes. In the third stage are included all cases in which the lesions are extensive and in which the lymph nodes show unmistakable evidence of metastasis. In 119 cases of cancer of the lower lip treated by roentgen rays, 86 were in the first, *22* in the second and 11 in the third stage. In the first and second stages there were 108 patients who did not show evidence of metastases at the time of treatment. Later, 4 patients developed malignant nodes in the neck, and died. The remaining 104 patients (or 96.3 per cent of the patients in the first and second stages) were well after five years. The 11 patients in the third stage had definite lymph node involvement at the time treatment was begun. All the patients in this stage died of cancer. It is the author's experience that early malignant lesions inside the mouth respond remarkably well to small amounts of radium in platinum needles implanted in the tissues. Interstitial radiation inside the mouth is augmented by highly filtered radium packs and roentgen rays applied externally. Oral hygiene is an important factor in the successful conduct of this class of cases. In incurable cancer of the mouth and lips, the patient can often be greatly benefited by radiation therapy, while in some cases life can be prolonged for months.

ADEQUATE TREATMENT OF SYPHILIS. E. T. BURKE, Lancet **1**:1127 (May 23) 1931.

Burke states that the main reasons why he adopted and teaches the alternating method of treatment for syphilis are: 1. Under the concurrent regimen, when arsphenamine and mercury were exhibited simultaneously, it was found necessary to interrupt treatment after the first eight or ten weeks in order to prevent the patient from showing signs of intolerance. An interval of from two to four weeks had to be allowed in which no therapy was given, so that the patient could recover

from the toxic effects of the remedial agents administered. During this time the parasite was permitted, unmolested, to entrench itself in deep-seated visceral structures, chiefly in the cardiovascular and central nervous systems. It therefore seemed more logical to try to arrange therapy so that rest periods were abolished and so that the parasite would be continuously subjected to an intense chemotherapeutic bombardment. This could be done only by exhibiting the drugs in alternate courses. 2. It was found that under concurrence there were many patients in whom jaundice developed. This was considered to be due probably to the double assault on the hepatic, renal and cutaneous systems. On changing over to the alternating system, the incidence of these complications, especially nephritis, almost reached the vanishing point. 3. Under the concurrent method, many patients were found to have become drug resistant or Wassermann-fast. This was thought to be due to the spirochete's having developed an immunity to the action of the remedial agents. It seemed natural, therefore, to reason that it would be better to administer the drugs in separate series so that no sooner did the parasite threaten to develop a resistance to one agent than a new weapon was brought into action, and when the efficacy of that drug began to wane, a change was made back to the original one or to another new one if one was available. Experience has shown this reasoning to be correct, for, since the adoption of the alternating method, drug resistance and Wassermann-fastness have ceased to appear as practical difficulties. 4. A study of the mode of action of arsphenamine and of bismuth (for mercury has been discarded as being inefficient and a mere waste of time) also forced the author to give up concurrent treatment. Arsphenamine and bismuth do not act directly on the parasite. When injected into the body they stimulate the tissues to produce toxalbumins (arsenoxyl and bismoxyl) that are lethal to Spirochaeta pallida. In other words, the arsphenamine and the bismuth are the raw materials supplied to the body factory that produces the finished article capable of destroying the parasites. The manufacture of these proteo-arsenic and proteo-bismuth combinations inevitably throws some strain on the producing tissues of the patient. When the two raw materials are exhibited simultaneously, twice as great a load is cast on the productive mechanism as when only one is given. The tissues are thus, under concurrence, more easily exhausted and may finally cease altogether to produce the lethal toxalbumins. This results in drug resistance and Wassermann-fastness. 5. Many patients who formerly had become Wassermann-fast under concurrence have been treated during the past ten years by the alternating method. A large proportion of the cases, as a result, became permanently negative. 6. Under the alternating method there is a saving in time and in the amount of remedial agents necessary to cure a patient with early syphilis.

TREATMENT OF SCLERODERMA BY PARATHYROIDECTOMY. R. LERICHE and A. JUNG, Bull. et mém. Soc. nat. de chir. **57**:609 (May 9) 1931.

Leriche and Jung performed parathyroidectomy on two patients with scleroderma, and after some preliminary considerations drew the following conclusions: It is indubitable that scleroderma is a surgical disease. It may be treated by various sympathetic operations or by a parathyroidectomy. At present the results obtained in employing the two methods are equal. The future will tell whether one should combine the two methods or prefer one to the other. Sympathectomy has already demonstrated its well established place and revealed the mechanism of the ailment. In serious cases of parathyroidectomy it has one advantage over sympathectomy. But it seems to invoke general as well as local changes afterward. By both methods one may now cure patients who have been considered incurable.

Society Transactions

MINNESOTA DERMATOLOGICAL SOCIETY

D. D. TURNACLIFF, M.D., *Secretary*

Meeting, April 1, 1931

C. A. BOREEN, M.D., *President, in the Chair*

A CASE FOR DIAGNOSIS. Presented by DR. H. N. KLEIN, St. Paul.

A woman, aged 71, was first seen about 6 weeks ago with cutaneous lesions of three months' duration. There were small miliary-like lesions in the mouth, especially around the gums and lower lip, which were edematous and painful to touch, and bled freely when the slightest friction was applied. The patient has had only ultraviolet therapy. Her condition was first thought to be miliary tuberculosis, but the results of an examination of the lungs were negative so far as active lesions were concerned.

DISCUSSION

DR. J. F. MADDEN, St. Paul: In view of the negative findings for tuberculosis and the associated bullae arising from apparently normal mucous membrane of the palate, a diagnosis of pemphigus must be considered. In order to rule out or to substantiate that diagnosis, I would send a specimen of blood to Dr. Pels for study, and also take a specimen of tissue for biopsy.

DR. H. E. MICHELSON, Minneapolis: I believe this to be a case of ulcerating miliary tuberculosis in spite of the negative findings in the x-ray photographs of the chest. The clinical characteristics are extensive ulceration, long duration without tendency to heal, miliary necrotic areas in the lesion and the ease with which the entire area bleeds. It is to be noted also that there is an ulcer at the base of the tongue, which is exceedingly painful. I do not believe that the condition is lupus vulgaris because of the absence of lupus nodules on the adjacent skin.

DR. S. E. SWEITZER, Minneapolis: One must consider two or three different conditions in this case: (1) tuberculosis ulcerosa acuta, a tuberculous ulceration that occurs as a terminal form of tuberculosis around the mucous orifices; this is free-bleeding, but with it one should find a very active tuberculosis in the lungs; (2) lupus vulgaris of the mucous membranes, which one usually finds in a much younger person. We have seen a few cases of lupus vulgaris starting later in life, however. I would suggest that tissue be taken for a biopsy, which might at least show whether it is lupus vulgaris. I believe the diagnosis lies between these two—tuberculosis ulcerosa acuta and lupus vulgaris.

DR. F. R. WRIGHT, Minneapolis: One interesting point is that this condition followed some dental work.

DR. J. F. MADDEN: That might possibly apply to the lesions on the lips, but how would you explain the vesicles appearing on the palate?

A CASE FOR DIAGNOSIS. Presented by DR. J. F. MADDEN, St. Paul.

J. W., aged 21, a student, first noticed an eruption on his face about two and one-half years ago while attending the University of Michigan. This persisted during the entire school year in spite of treatment. On returning to his home in Colorado during the summer, the eruption disappeared. He entered the University of Minnesota last year, and the eruption recurred.

At this time the patient presents an eruption confined to the face, neck, flexor surfaces of the arms and medial surfaces of the thighs. This presents itself as brightly inflammatory patches and plaques varying in size from that of a dime to that of a silver dollar. The lesions exhibit central clearing, peripheral spread and pinhead-sized inflammatory vesicles at the borders. The skin of the entire face is thickened and erythematous. The patient was presented at his request and for therapeutic suggestions.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: The distribution of the eruption over the exposed surfaces, with the seasonal variations, points to a dermatitis from external irritants. The attacks coincide with his attendance at law school, and it may be that the books themselves, the covers or the glue, may be at fault. Local application to the skin can be expected to give only temporary relief, unless at the same time the boy is separated from the external irritating factor.

DR. T. F. MADDEN: I have treated this patient at intervals for two and a half years. All the sensitization tests were done two years ago, but none was positive. The tests were repeated this year, and the patient exhibited a positive reaction to goat hair.

A CASE FOR DIAGNOSIS (QUESTIONABLE SYPHILIS?). Presented by DR. E. C. GAGER, St. Paul.

This woman presents annular lesions on the forearms and on the back. The lesions are a dull red and are slightly raised or infiltrated on the borders; the centers of the lesions are apparently normal. On the back, the eruption forms large gyrate figures, some concentric figures and a general annular outline. There are no symptoms, and the condition has been present for a number of years. The Wassermann reaction of the serum and that of the spinal fluid are negative.

DISCUSSION

DR. H. E. MICHELSON: My diagnosis is erythema annulare centrifugum. The border, the lack of exudation, the ringed eruption making fantastic figures and the rubbery consistency are the characteristics on which I base my diagnosis. This is the second case of this kind, and you will recall that in our former case the question of syphilis was discussed, but ruled out. In regard to the annular macular recidive, the so-called neurorecidive of Unna, this type of syphilitic eruption is very rare, occurring almost invariably in patients who have previously had insufficient treatment, and is characterized by large rings that are not palpable. I have had one such case, verified by Dr. Sweitzer, and it required about three months of active treatment to cause a resolution when the patient had had a year of previous treatment. There is no therapy for erythema annulare centrifugum, but the condition usually disappears of its own accord.

DR. C. A. BOREEN: I saw that case at the dispensary two or three years ago. At first we thought it was tinea, but went into it very thoroughly and had cultures made. It resembled tinea imbricata. Syphilis was also considered.

DR. E. C. GAGER, St. Paul: Stokes refers only very briefly to the condition known as neurosyphilid by Unna, and these lesions on the arms are very suggestive of it. Dr. Fox described one of these cases of macular annular syphilis, and Fournier described a number of them. The French call it erythema tertiare. It is a later tertiary manifestation and lasts for years. That is the condition we will have to rule out in this case. This woman had a positive Wassermann reaction some time ago, but a recent Wassermann reaction was negative. Biopsy ought to be of great aid. The spinal fluid is normal. I am inclined to think it is syphilis, and that it belongs in that classification. I happened to be in Schamberg's clinic in Philadelphia when a patient with this type of macular syphilid came in, who had just developed this recidive. Dr. Schamberg says that these macular annular syphilids are becoming more common, and he thinks that insufficient treatment with neoarsphenamine has something to do with it. If this is syphilid, it is a form

different from that of the ordinary syphilid that we see. These cases have been reported as clinical entities, as a different type of syphilis. My interest in these cases is to determine whether or not they are that type.

DR. L. A. BRUNSTING: No doubt tissue will be taken for a biopsy and further study made of the question of syphilis, including a therapeutic test over a period of several months. The configuration of the lesions and the occurrence of new lesions within the areas previously affected, together with the lack of atrophy, are decidedly unusual.

ACRODERMATITIS ATROPHICANS. Presented by DR. E. C. GAGER, St. Paul.

A white woman, aged 67, entered the hospital with ulcers of the right leg in the region of the ankle. These ulcers healed after two weeks of rest in bed. The skin of both legs is red and shiny, very thin and atrophic. There is a sharp line of demarcation in the upper third of the thigh, above which there is normal skin. The condition has been present for fifteen years or more, and it is remarkable that the patient has recently had an exacerbation of the dermatitis, showing that the process is still active. She is otherwise well. The laboratory findings are negative.

DISSEMINATED LUPUS ERYTHEMATOSUS ACUTUS. Presented by DR. C. D. FREEMAN, St. Paul.

J. T., a man, aged 50, first noticed a discoloration on his forehead about two years ago. This progressed until the jaw, neck and hands became involved. The patient has felt well up to this time. At present the eruption is a dusky red, and involves the jaw, neck and hands. The involved skin is dry and atrophic in patches, and presents some telangiectasia and a tightly adherent dry scale. There are marked follicular dilatation and plugging. The results of a biopsy are consistent with lupus erythematosus.

DISCUSSION

DR. H. E. MICHELSON: I believe this to be lupus erythematosus in extenso, not acute disseminated lupus. I think the patient should be observed for a long time, if possible, because there are many factors that make me think that in this patient a poikiloderma atrophicans vasculare may eventually develop. It will be recalled that when Jacobi demonstrated his first case of this condition before the German Dermatological Society, Professor Jadassohn and, I believe, Professor Arndt thought that the condition closely resembled an extensive lupus erythematosus.

DR. L. A. BRUNSTING: The entire face is glazed and of a bronze hue, with erythema sharply demarcated over the upper part of the forehead. However, the lesions about the hands and forearms make a diagnosis of subacute disseminated lupus erythematosus most likely. The general state of well-being of the patient belies the activity of the lupus process.

PHILADELPHIA DERMATOLOGICAL SOCIETY

J. P. GUEQUIERRE, M.D., *Secretary*

April 3, 1931

C. S. WRIGHT, M.D., *President*

A CASE OF SENSITIVENESS TO MERCURY. Presented by DR. F. D. WEIDMAN for DR. J. B. LUDY.

There is nothing curious about the case presented except that there was a flare-up in connection with two different preparations of mercury. R. J., 24 years of age, had pediculosis pubis; he treated himself with blue ointment procured at a drug store, and the parasites disappeared. About three or four weeks after the first attack, the patient complained of itching. He then bought mild mercurous

chloride from a druggist, using it as a dusting powder on the skin; following that treatment a dermatitis developed which is seen now. It is folliculitis extending from the inguinal region down to the inside of the thighs and buttocks—a common picture of postmercurial dermatitis. The interesting feature of the case is that the dermatitis appears to have been provoked by the application of mild mercurous chloride, which ordinarily does not cause this reaction; but in view of the preceding application of mercury in another form, the question of sensitization is raised—not of course a protein allergy, but a chemical sensitization.

DISCUSSION

DR. R. GILMAN: I agree with Dr. Weidman that this is a case of sensitiveness to mercury, especially in view of the fact that the second medicament applied was mild mercurous chloride. Dr. Weidman told me that the second application was confined to the inguinal region and did not include the buttocks, where the patient has dermatitis. It seems worth while to try the patch test in this case.

DR. A. STRAUSS: We have used a great deal of 50 per cent mild mercurous chloride ointment, and we rarely see dermatitis following an application of this strength.

DR. E. F. CORSON: The fact that both mercurial preparations were used on the same area confuses the outcome a little. If the patch test were used on some distant part of the body, such as the back, it might throw a little more light on the case. In our experience mild mercurous chloride has been a fairly inert external medicament.

DR. F. D. WEIDMAN: I have seen one case in which ammoniated mercury caused a severe folliculitis; the preparation had been prescribed for tinea cruris by a medical student.

DR. C. S. WRIGHT: In reviewing the literature on mercurial eruptions some years ago, I found the case of a man who died after a single therapeutic dose of mild mercurous chloride by mouth. Cases of extreme susceptibility to mercury are not extremely rare. Some years ago I was called to see a man in North Philadelphia who had applied blue ointment only once to the groins for treatment for pediculosis pubis. The external genitalia were enormously swollen, and the surrounding skin was bright red and edematous; in fact, the skin of the entire body was markedly reddened. He was suffering from severe toxemia and a mild degree of nephritis. Fortunately, under the influence of starch baths and alkalinization he recovered.

A CASE OF LICHEN SCROFULOSORUM TREATED WITH GOLD SODIUM CHLORIDE INTRAVENOUSLY. Presented by DR. S. S. GREENBAUM.

G. F., a man, aged 42, who was first seen by me on Feb. 9, 1931, stated that he had had a generalized eruption since September, about five months. He had a sudden fall of hair about the same time that the eruption on the body developed, and the eruption did not itch. On examination I found a number of irregularly rounded patches that consisted of pinhead size, discrete, follicular papules, some pink, some skin-colored and some armed with a hyperkeratotic lance not unlike what one sees in ordinary lichen spinulosus. A good deal of the eruption has disappeared since February 9 under treatment with intravenous injections of gold and sodium chloride. The first two doses were 1 mg. each, the last seven, 2 mg. each. There were a cicatricial patch of alopecia on the scalp and several patches of alopecia areata. The rash improved 90 per cent in the four weeks before presentation. I could not account for the cicatricial loss of hair which the patient said occurred suddenly. All studies of the blood and urine gave negative results. The patient was seen at the university, and Dr. Ireland can tell you about the findings.

DISCUSSION

DR. F. A. IRELAND: We examined stumps of the hair for fungi with negative results.

DR. F. D. WEIDMAN: The condition looked somewhat like pityriasis rubra pilaris, but neither Dr. Strauss nor I could see any corroborative evidence of that on the backs of the fingers. The patch on the scalp I think is ringworm. I am sure that I saw two or three very crooked hairs there that could qualify as stumps. Trichophyton violaceum is a particular form that often produces a condition simulating favus, which can continue on beyond puberty and is not spontaneously regressive as are most of the other forms of ringworm. I should not be surprised if Trichophyton violaceum were isolated from these lesions if the appropriate hairs were examined.

DR. JAY F. SCHAMBERG: This case is a very interesting one in view of the association of two conditions that are entirely different in character and appearance. I do not believe that the condition on the scalp is ringworm, for the following reasons: In the first place, ringworm in a man of this age is exceedingly rare. I have never seen ringworm in a man as old as this patient. In the second place, he had had a diffuse inflammatory condition of the scalp which still shows traces of redness and friable crusts, and over this entire area there is a pronounced atrophy. I believe that he had an acute lupus erythematosus on the scalp. It is difficult to say what the condition on the body is, because a considerable amount of involution has taken place. The patient has a widespread pinhead-sized papular eruption closely aggregated in some of the patches, and one might consider the possibility of the following conditions: lichen spinulosus, lichen scrofulosorum and pityriasis rubra pilaris or its equivalent, lichen ruber acuminatus. Lichen ruber acuminatus is believed at times to be of tuberculous origin, as is, of course, lichen scrofulosorum. Whether one could assume that there is lichen ruber acuminatus associated with lupus erythematosus of the scalp and bring forth convincing evidence is another matter. The patient does not present what we commonly find in lichen ruber acuminatus, prominent papular elevations on the dorsal surfaces of the phalanges and the back of the neck, so that I think a positive diagnosis of the condition on the body cannot be made today. The fact that his condition has improved very greatly with the treatment with gold compounds lends some color to the thought that the whole condition may be of tuberculous origin, although, of course, there is a considerable discordance of opinion as to the origin of lupus erythematosus.

A CASE OF PITYRIASIS LICHENOIDES ET VARIOLIFORMIS ACUTA. Presented by DR. SIGMUND S. GREENBAUM.

A man, aged 37, about eight weeks before presentation developed an eruption that was preceded by general malaise. The eruption began in the belt region, and more lesions appeared every day until the number seen at present had developed, in about fourteen days. He was seen three weeks after the appearance of the eruption by a physician who reported him to the department of health, and the inspector of the department made the diagnosis of varicella. I saw him after he had been in quarantine for about three weeks, and disagreed with the diagnosis. He presents practically the same condition now as he did then: a generalized, profuse eruption that has spared the face, the scalp and the mouth. The eruption consisted of macules and pink, discrete papules varying in size from that of a pinhead to that of a pea, some with and some without scales and some covered with hemorrhagic crusts. As the lesions disappeared, the papules flattened, and as the scales dropped off, the papules had a shiny appearance, not unlike that of the papules in lichen planus. On disappearing lesions, the scales were easily detachable, but on earlier lesions their removal left a bloody ooze. Some superficial, depressed

scars, the size of a pinhead, were present here and there. A half dozen pustules, varying in size from that of pinpoint to that of a pinhead, were likewise present, and there was one pustular lesion on the left palm. When the quarantine was lifted, the patient was asked to come to the Graduate Hospital, where Dr. Schamberg, Dr. Wright and I examined him. The diagnosis was a drug eruption or parapsoriasis.

The histologist's report was that the papule removed several days previously showed on section a very small amount of inflammatory exudate in the corium, that it was not a tumor, and that the picture was not that of tuberculosis or syphilis. The Wassermann report was not received before presentation of the patient.

NOTE.—The Wassermann and Kahn tests both gave a reaction of 4 plus, and the only history of genital disturbance was that the patient had gonorrhea twelve years before presentation.

The eruption did not change much during the two weeks subsequent to presentation. The patient was given a mixed treatment and also received an injection of 0.3 Gm. of neoarsphenamine immediately after presentation and 0.06 Gm. of neoarsphenamine seven days later.

DISCUSSION

DR. F. D. WEIDMAN: I do not think we ought to let important cases like this go by without additional comment, but at the same time I do not know what to say, because I am not familiar with this particular condition as diagnosed. On superficial examination, the condition appears to be an extremely acute kind of psoriasis, with some rather deeply infiltrated lesions covered by fine scales; there is nothing to support that diagnosis in the way of distribution. In important places, as in the axillae, there are lesions that are entirely too wet. I think we should all be very much interested in seeing this patient again after a number of weeks to see what has subsequently developed. It is a matter of information and education to learn how an unusual case like this will turn out.

DR. J. F. SCHAMBERG: This is one of the most interesting cases that I have seen in a long time. I saw the patient about a week ago, and there is not much change in his appearance since then. I agree with Dr. Greenbaum in the diagnosis. I think the condition belongs in the group of parapsoriasis, but it exhibits marked deviations from the ordinary type of case. The patient has thousands of guttate, reddish lesions over the body; some of them began as vesiculopustules. The first time I saw him he had a number of distinct crusts on the lesions, which were due to drying of fluid. The eruption is gradually undergoing a change in appearance. On the shoulders and neck some of the lesions are taking on a lichenoid appearance, and I think the probability is that this condition belongs in the same class as pityriasis lichenoides chronica. I saw such a case today for the third time. The patient was sent to me by his physician, who believed that the condition was syphilis, and I may add that the patient presented here would probably be regarded as having syphilis by the vast majority of general practitioners whom he might consult. His physician refused to believe that the condition was pityriasis lichenoides chronica; he gave him several medium-sized doses of arsphenamine, and thought that the eruption was improving. He made provocative Wassermann tests after that (he has now made eleven Wassermann tests, all negative), and I can see no material change in the eruption.

These cases are of particular interest and importance from a diagnostic standpoint because of the liability of confusion with syphilis. Of course, there is some resemblance to other dermatoses also, but I do not recall that I have ever seen another case exactly like this.

A Case of Morphea. Presented by Dr. J. F. Schamberg and Dr. C. S. Wright.

Mrs. R. R., aged 33, stated that she had had patches on her leg for three years. They appeared as small reddish lesions, which gradually extended peripherally; at the same time a new one occasionally developed, until there were about four patches. These varied in size from that of a small orange to that of a man's palm; they had a reddish tint, the centers were atrophic, and when one pressed out the redness the skin had a waxy appearance. On the occasion of the patient's first visit, palpation of the lesions revealed a definite boardlike feel; there was an elevated border, but this has partially disappeared during the past month. She has had no treatment except a simple emollient locally and about four treatments with the high frequency current. She took an Asiatic pill twice daily. Although we believe the case to be one of morphea, we present the patient for opinions as to the diagnosis.

She was previously seen at the University Hospital clinic but was frightened away by hearing the word cancer used in connection with these lesions. A biopsy was made at the University Hospital, and I shall ask Dr. Ireland to give us a report of their findings.

DISCUSSION

Dr. F. A. Ireland: There was considerable thinning of the epidermis. There was marked increase in the fibrous tissue in the corium, and it was quite vascular—there were a number of blood vessels in the corium.

Dr. A. G. Schoch: There was no evidence of central infiltration or an inflammatory process of any kind. Was the condition morphea or epithelioma or was it some other condition? Biopsy proved that it was not epithelioma. There was some increase of collagen in the subepithelial layers.

Dr. S. S. Greenbaum: I was wondering whether one could not consider this condition Majocchi purpura rather than morphea. The lesions are dark red and symmetrically disposed, and the centers are atrophic and alopecic.

Dr. F. D. Weidman: There does not seem to be much stiffening of the skin; it seems to be soft and pliable. On pressure there is no difficulty in obliterating the red coloration (it could not be expressed if it were hemorrhage); one can produce an ivory color, and I think that the yellow factor is a result of hyperplasia and degeneration of the elastic tissue. This change is rather common in old scars, morphea, lupus erythematosus, etc. In this case there is not old scar tissue but merely elastic tissue hyperplasia.

Dr. J. F. Schamberg: I agree with Dr. Wright in the diagnosis of morphea, although the condition is a very unusual type of morphea. There are the two large patches on the leg, which show distinct evidence of atrophy; not only marked thinning of the epidermis, as is evident by sight and by tactile sense, but complete atrophy of the hair follicles. You observed that the patient has considerable large, coarse, dark hair around the patches, but the hair on the patches has entirely disappeared, and there are no hair follicles visible. There is pronounced telangiectasia all through the patches, and the skin is more dense to the touch than the surrounding skin and less elastic, so that there is the atrophic stage of a morphea that is unusual on account of the remarkable glossy appearance of the surface and the redness due to the large blood vessels coursing through the superficial portion of the skin. It is difficult to say anything regarding the origin of this condition, but I have seen patches of morphea that were unquestionably due to local injury of a nerve, as when a woman falls and an ecchymosis develops on the arm or leg; this is followed by some nerve change, and then a patch of morphea develops. The two patches in this case are remarkably symmetrical; it is difficult to assume that a similar injury occurred on both sides, but I think that possibility must be kept in mind. The condition is distinctly different, however, from the ordinary type of morphea.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

A. BENSON CANNON, M.D., *Secretary*

Regular Meeting, April 7, 1931

JOSEPH J. ELLER, M.D., *Chairman*

A CASE FOR DIAGNOSIS (ERYTHEMA MULTIFORME?). Presented by DR. HERMAN GOODMAN.

A woman visited the clinic of the New York Skin and Cancer Hospital on March 7, 1931, complaining of an eruption, reddish, rather symmetrical in its distribution and having a tendency to assume ring shape. The eruption was confined to the arms, neck and face. On the lower left eyelid was a rather flattened, yellowish-brown plaque, which had been previously treated. On the conjunctiva of the left eye, beneath the lower eyelid, the patient presented a small tumor.

The trouble had started two and a half years prior to presentation, with a swelling in the right leg, diagnosed as phlebitis, for which the patient had remained in bed for seven weeks. About two months later, she noticed a purplish flush of the neck and face, which shifted from place to place (not nodular at that time). There was no itching. About the same time, a large purplish patch appeared around the left eye. As the color disappeared, a nodule appeared on the lower eyelid. This lasted three months, and the conjunctival lesion developed. The condition of the skin as a whole remained about the same until six months prior to presentation; then the skin became more infiltrated and began to itch. The skin on the body and lower extremities remained clear.

In Africa, the patient had received neoarsphenamine for five years (1926-1931) as a precautionary measure against yaws. There diarrhea developed, and emetine (twenty-one injections) was given, also serum two or three times from 1926 through 1930. From 1927 to 1929, the patient had malaria. Seven years prior to presentation, both tubes and one ovary were removed. In 1926, menstruation stopped. The condition of the skin appeared six months after the patient began using bromides. She continued using bromides off and on for one year after the trouble appeared, but it did not tend to make the condition worse.

The results of a complete examination of the blood and of urinalysis were negative. The Wassermann reaction of the blood was likewise negative.

NOTE.—Subsequent to the presentation, a specimen was offered for biopsy. The microscopic diagnosis was erythema multiforme.

DISCUSSION

DR. GEORGE MILLER MACKEE: I regret my inability to make a clinical diagnosis. It is not a suitable case for immediate clinical diagnosis. It must be worked up. Erythema multiforme perstans is the probable diagnosis. Dermatitis medicamentosa must be carefully ruled out. Even the possibility of leukemid must be considered. The condition of the eyelid is xanthoma. The lesion of the conjunctiva may be a keloid. I have seen several keloids of the mucous membranes following pyodermia.

DR. FRED WISE: The appearance of the lesions on the forearms is very suggestive of amyloid degeneration of the skin.

DR. SIGMUND POLLITZER: This is a case that requires more extensive study than it is possible to give in the few moments at our disposal. I have been thinking of the possibility that the lesion on the arms is the result of a metabolic distur-

bance in which there is a deposit analogous to that in the xanthomatous lesion on the eyelid. I think Dr. Wise had something of the same thing in mind in speaking of amyloid degeneration. Obviously, a diagnosis can be made only with the aid of the microscope and the necessary chemical tests in addition.

DR. GEORGE MILLER MACKEE: It will be well to settle at once the question of the duration of individual lesions. I understand that the individual lesions endure for some time and then disappear. That seems to me to rule out the two conditions mentioned by the last speaker.

DR. HERMAN GOODMAN: As far as I can recall from the patient's statement, the lesions are not permanent.

DR. FRED WISE: The patient states that the lesions on the forearm were of three years' duration.

DR. JOSEPH J. ELLER: The patient told me that the lesions had receded somewhat, but never had disappeared.

PEMPHIGUS. Presented by DR. EUGENE TRAUB.

The patient entered the clinic on Oct. 14, 1930, with a bullous eruption limited to the temporal region. The condition had been recurrent and had been present for one year. All forms of medical treatment had failed to bring an improvement. The general examination revealed nothing of interest. The results of a complete examination of the blood were normal. The microscopic diagnosis was pemphigus.

For two months the patient showed marked improvement from six one-fourth unit roentgen treatments weekly. The bullous condition then recurred. The patient had previously been presented before the New York Dermatological Society, in February, 1931.

DISCUSSION

DR. MAX SCHEER: I hesitate to make a diagnosis from only two lesions. I would rather keep the patient under further observation.

DR. GEORGE MILLER MACKEE: I do not believe it is possible to make an unequivocal diagnosis in this case. I have seen the patient twice. It is logical to make a diagnosis of probable pemphigus, but in order to substantiate that diagnosis, it may be necessary to observe the patient for a period of years. Pemphigus may occur in a localized area, the mouth, for instance, for many years before a generalized eruption appears. I understand that the result of the phytopharmalogic test was positive. While by no means infallible, a high reading is suggestive of pemphigus. Other diagnoses that have been considered are a bullous type of lupus erythematosus and also recurrent herpes.

DR. SIGMUND POLLITZER: I agree entirely with Dr. MacKee. One cannot make a definite diagnosis of pemphigus; one must await developments. Pemphigus often begins with a few lesions in a limited area, coming and going for a long time, and then suddenly becomes widespread and presents the characteristic picture. It is enough to have thought of the possibility of pemphigus in this case.

DR. JOSEPH J. ELLER: Would any one consider it significant that the specimen taken for biopsy looked microscopically like pemphigus?

DR. EUGENE TRAUB: A sample of blood was sent to Dr. Pels, who reported that it was toxic, suggesting that the diagnosis of pemphigus is correct. This patient's appearance changes from week to week so much that it is difficult to know exactly what he may show when presented at a meeting. Tonight he has fewer vesicles and blebs than usual, but they come in successive crops, and I have seen him with these scattered well over the face, although for a long period they were limited to the area about the temples. Blebs have appeared on the eyelids and margins of the lid. None has been noted on the mucous membranes of the mouth. An interesting point is that at a previous presentation everyone agreed that there was definite atrophy in the region of both temples, where the lesions have been

most pronounced. This led to the suggestion that the condition might be a bullous type of lupus erythematosus. It was for this reason that a biopsy was performed, and our pathologist suggested that from the section pemphigus was the more likely. Strange to say, the apparent atrophy is not nearly so pronounced at present. The eruption has been present continuously for almost eighteen months, but the patient had a previous attack twelve years ago, lasting about a year.

ERYTHEMA ANNULARE CENTRIFUGE. Presented by DR. BINFORD THRONE and DR. JEROME KINGSBURY.

D. G., aged 42, was first seen in the clinic on Aug. 27, 1930. The duration of the eruption at that time had been about one year. It had always been limited to its present site on the back of the neck. The lesions consisted of papules, papulonodules and erythematous plaques, with a tendency to be arranged in circles.

The results of the urinalysis were negative. Two Wassermann tests of the blood gave negative results. Chemical analysis of the blood showed: sodium chloride, 446 mg. per hundred cubic centimeters; sugar, 111.2 mg.; urea nitrogen, 11.5 mg.; uric acid, 3.08 mg., and carbon dioxide, 63.7 per cent by volume.

Treatment consisted in the administration at various times of rhubarb and soda, mercury and iodides by mouth, and the following drugs intravenously: calcium chloride, sodium thiosulphate and methenamine. He also received a series of roentgen treatments locally in fractional doses.

DISCUSSION

DR. CHARLES WOLF: I saw this patient about a year and a half ago, at which time he presented a unilateral eruption located on the back of the neck on the right side. The eruption at that time consisted of infiltrated papules, which coalesced, forming circinate patches with clearing centers. I made a diagnosis at that time of erythema multiforme perstans because of the persistence of the lesions under various types of treatment, i. e., x-rays, ultraviolet rays, etc. At present the eruption has spread to the other side of the neck and has not the characteristic circinate form, but is more of a diffuse papular variety without any of the circinate arrangement.

DR. BINFORD THRONE: As the history states, the patient has been under observation since last August. During that time we have had opportunities to observe the changes that have come in the lesions. At times, they have resembled almost an annular papular syphilid; at other times, the erythematous element has been more marked. The condition, I think, corresponds clearly to that described by Darier as erythema annulare centrifuge.

A CASE FOR DIAGNOSIS (ERYTHROSE PERIBUCCALE PIGMENTOSA?). Presented by DR. HERMANN FEIT.

M. K., aged 21, born in Czechoslovakia, a waitress, had always been in good health. A year prior to presentation, she had first noticed that the skin on her forehead had become brown. Gradually these brown spots spread over the middle of the face. Menstruation was normal. Besides mild acne, there had been no other findings of importance. There was a poorly defined hyperpigmented patch occupying the center of the face.

DISCUSSION

DR. JOSEPH J. ELLER: This might be chloasma.

DR. SIGMUND POLLITZER: It corresponds very well to Brocq's description, even if not in every detail, and I think the diagnosis is correct.

DR. HERMANN FEIT: At first I thought of chloasma uterinum. As the pigmentation, however, is present only in the midface, I was struck by the similarity to the case presented by Dr. Ormsby under the aforesaid title, although I do not remember having seen a case before.

PITYRIASIS RUBRA PILARIS. Presented by DR. EUGENE TRAUB.

S. K., a Russian, aged 65, presented a redness and scaliness of the skin of the scalp, face, neck, chest, arms, abdomen and thighs and an ectropion of the lower eyelids. The condition had begun six months prior to his presentation, with a scaly, itchy patch on the abdomen, and within the following three weeks had become generalized. There had been no previous skin trouble. The patient had always been in good health, except for high blood pressure for fifteen years.

DISCUSSION

DR. PAUL BECHET: The eruption shows a predilection for the characteristic location of seborrheic dermatitis. The scalp is markedly involved. The lesions are extremely scaly, without frank follicular hyperkeratosis, even on the backs of the fingers. The palmar keratosis is not unlike that usually observed in exfoliative dermatitis. I should seriously consider the diagnosis of secondary exfoliative dermatitis on a primary seborrheic dermatitis, in preference to pityriasis rubra pilaris.

DR. FRED WISE: In spite of the absence of hyperkeratotic lesions on the dorsa of the phalanges, I believe that the other features of the eruption, taken as a whole, justify the diagnosis as presented.

DR. SIGMUND POLLITZER: I am much in doubt about this case. The only thing in my mind that speaks for pityriasis rubra pilaris is the presence of the follicular plugs in a few patches on the abdomen, especially on the side. The characteristic follicular lesions on the terminal phalanges of the fingers are absent. That does not rule out pityriasis rubra pilaris, but, in view of the fact that the whole picture is that of an exfoliative dermatitis, which might be due to a number of causes, the case might be grouped as any one of a number of diseases in which that is the main symptom. I would put down any diagnosis with a question mark. I do not think we can label this definitely pityriasis rubra pilaris.

DR. GEORGE MILLER MACKEE: The patient demonstrates one feature that is often seen in pityriasis rubra pilaris, i. e., the sharply outlined clear areas of skin in the midst of sheets of eruption. That, with the follicular papules of the trunk, the possible beginning keratotic plugs on the backs of the fingers, the palmar and plantar keratoses and the large sheets of eruption, leads me to agree with the diagnosis as presented. As Dr. Pollitzer said, there are other conditions that could produce an eruption simulating pityriasis rubra pilaris. Arsenical dermatitis is one.

DR. BOLESLAW LAPOWSKI: In mycosis fungoides and parapsoriasis we often see sharply defined areas of normal skin. This feature is not pathognomonic of pityriasis rubra pilaris.

DR. BINFORD THRONE: The possibility of an arsenical dermatitis brought up by Dr. MacKee must be taken into consideration. I hardly think that condition would occur in these circumscribed patches. We had a case similar to this one at the New York Skin and Cancer Hospital recently; lead was found in the urine. My diagnosis is pityriasis rubra pilaris.

DR. LOUIS CHARGIN: I am inclined to believe it is pityriasis rubra pilaris. A point in favor of this is the keratosis on the palms, which is characteristic of this disease. There is perhaps more scaling than is ordinarily observed, but, aside from that, I see no reason to doubt the diagnosis. The seborrheic condition on the scalp is also frequently associated with pityriasis rubra pilaris.

DR. EUGENE TRAUB: In addition to the many features that have already been mentioned by Dr. MacKee and others, the marked ectropion of both lower eyelids that has resulted in so short a time seems to me another feature confirming the diagnosis. Of course, I realize that ectropion of this character is seen in other conditions of the skin, but usually only when they have been of much longer standing.

A Case for Diagnosis (Epithelioma?). Presented by Dr. John H. Harris.

When first seen, on April 6, 1931, the patient, aged 57, presented a fungating lesion, 2 cm. in diameter, located in and about the soft commissure, involving the buccal mucosa for approximately 2 cm. The borders of the lesion were sharply defined, raised and rolled, with a gelatinous appearance. The patient stated that the lesion had been present for twenty years and for five years had been stationary in size. There were no palpable glands. The results of both Wassermann and Klein tests were negative.

DISCUSSION

Dr. Edward R. Maloney: The duration of this lesion for twenty-two years tends to rule out the diagnosis of carcinoma or tuberculosis. To me it seems to be a nonmalignant chronic granuloma of some kind. As to causation, the patient has a good explanation that he has formulated himself. He says that for the past twenty-two years or more he has had a marked hyperacidity of the stomach, accompanied with vomiting and regurgitation of acid fluid. At night he sleeps with his head on the side on which the growth is, and there has been a more or less constant dribbling of irritating fluid over that corner of the mouth. This condition has been greatly improved by treatment during the past year, and as a result the growth is gradually diminishing in size.

Dr. George Miller MacKee: Tuberculosis should be considered.

Dr. Fred Wise: If the lesion has existed for twenty-two years without palpable glands in the neck, I think one can rule out carcinoma. The buccal lesion closely resembles tuberculosis, as suggested by Dr. MacKee.

Dr. Louis Chargin: I suggest a common wart. A biopsy ought to be done in order to make a definite diagnosis.

Dr. Max Scheer: I quite agree with Dr. Chargin that the lesion may be a hypertrophic wart.

Dr. Sigmund Pollitzer: I am inclined to view the growth as malignant, notwithstanding its twenty-two years' duration. There is no law that a malignant growth must advance within any definite time. I base that diagnosis not only on the appearance of the lesion, but on its hardness. There is a conflict of testimony on the point of glandular involvement. The man certainly has a palpable hard gland in the submaxillary region. Instead of taking a specimen for biopsy, I would suggest taking out the entire lesion, a radical operation. I think the gland should be removed at the same time.

Dr. Boleslaw Lapowski: If the man has had the growth for twenty-two years, why interfere?

Dr. Sigmund Pollitzer: In twenty-two years and two months there may be general metastases.

Dr. J. Frank Fraser: I am interested in this case. The question of carcinoma should be considered, and that of tuberculosis must also be considered, and as Dr. Chargin suggested, a diagnosis of verruca is possible. The history is against prickle cell epithelioma. It is unusual for that type to run a course of twenty years.

Replying to Dr. Goodman's question, why it should not be a carcinoma of the basal cell type, the so-called basal cell epitheliomas never arise in mucous membranes. It is generally agreed among pathologists that these tumors arise from a group of cells that under normal development are destined to form the hair matrix, and as there are no hairs in the buccal membrane, we can rule out basal cell carcinoma.

The enlarged lymph node in the neck may be due to an inflammatory process. Whether the condition is carcinoma, tuberculosis or plain verruca can be settled only by microscopic examination. It is in my opinion a safe procedure to do a biopsy in these cases.

DR. MARION B. SULZBERGER: I agree with all that has been said concerning the clinical appearance of the lesion. In view of this fact, I think it is possible to consider something else, i. e., a compromise between these diagnoses. We have a verrucous lesion of the mucous membrane which in some ways resembles a chronic granuloma and in other ways a carcinoma. There are certain lesions of the mucous membranes that are precancerous, and that are clinically like this, i. e., present some elements of a verrucous lesion, of a granuloma and of a carcinoma. The condition might be Bowen's disease of the mucous membranes. Biopsy would show that. I should like to hear what Dr. Eller has to say.

DR. JOSEPH J. ELLER: I lean toward the diagnosis of tuberculosis.

DR. LOUIS TULIPAN: I had the impression that this is a primary tuberculosis. Ulcerative conditions of the mucous membrane of the mouth are invariably secondary to pulmonary tuberculosis, and one has to examine the chest to see if there is an active condition there. I believe it is primary if it is tuberculosis.

DR. JOHN H. HARRIS: I do not agree with the diagnosis of tuberculosis because I do not know of any form of tuberculosis that can be present for twenty years without causing more destruction of tissue than we have here. In spite of the duration of 20 years, without any adenopathy, I still believe this growth to be carcinoma.

NOTE.—A microscopic examination made subsequent to the meeting showed, in the opinion of one pathologist, squamous cell epithelioma. The sections, as yet, had not been submitted to others for an opinion.

SYPHILIS (EARLY OR LATE?). Presented by DR. VAN ALSTYNE CORNELL.

R. L., a man, aged 36, born in Canada, came to the New York Post-Graduate Hospital Clinic on March 11, 1931. On the outside of the right arm near the elbow was a dollar-sized ulcer with a raised, crusted border, the floor of which was covered with a bloody, thick, grayish serum. The lesion had begun three or four weeks before as a small papule. On the left cheek there was a dime-sized, raised, crusted lesion.

At the time of his admittance mycotic infection was thought of, but examinations for tinea yielded negative results. There was no adenopathy. Two dark-field examinations revealed nothing. The result of the Wassermann examination was 4 plus. The lesion improved considerably with applications of mild mercurial ointment.

DISCUSSION

DR. ISIDORE ROSEN: I saw this patient at the clinic on his first visit. At that time the lesion on the elbow, although superficial, was sharply outlined, and I suggested a dark-field examination for Spirochaeta pallida to rule out the possibility of a primary lesion. In view of the rupial lesion on the face, I am of the opinion that the ulcer of the elbow is an unusual form of chancre in a person with lowered bodily resistance.

DR. RAY H. RULISON: I think the patient has early syphilis and extremely poor resistance to the disease.

DR. EUGENE TRAUB: This patient has a round lesion, at one portion of the border of which is an irregularly shaped area that suggests the possibility of a local application at this site. As the configuration of the lesion is not of the type usual in late syphilis, I suggest the possibility of an artefact in a patient with a positive Wassermann reaction.

DR. ELIAS ABRAMOWITZ: When this man first presented himself at the Post-Graduate Hospital about three weeks ago, only the ulcer on the arm was noted. No tinea was found on microscopic examination, and a dark-field examination revealed no spirochetes. The ulcer was suspected of being an artefact. When about two weeks later he reappeared at the clinic, it was noted that he had a rupioid lesion on the left cheek and also on the back of his hand. The ulcer

on the arm had increased in size. A weak mercurial ointment was ordered for the ulcer on the arm and a Wassermann test was made. The reaction was reported to be 4 plus. The use of the blue ointment for three days had caused almost 50 per cent improvement in the condition. The man stated that he had had two attacks of gonorrhea many years before, but that he had not had chancre or other eruptions of the skin. He appeared undernourished and pale.

DR. J. FRANK FRASER: Has the patient been taking iodides?

DR. VAN ALSTYNE CORNELL: There was no history of ingestion of drugs. The secondary ulceration of which Dr. Traub speaks I think is due to the taking of a specimen for biopsy, the result of which has not yet been reported. A diagnosis of artefact was made when the patient first came in. A mycotic lesion was also thought of. However, examinations did not show any tinea. Antisyphilitic treatment has not yet been started. We did not get the result of the Wassermann test until yesterday.

NOTE.—The histologic examination was made subsequent to the meeting. The diagnosis was gumma.

ESPUNDIA AND CUTANEOUS MYIASIS. Presented by DR. GEORGE C. ANDREWS.

X, a botanist, aged 26, stated that from June, 1930, to January, 1931, he had been in Honduras on a botanical survey, and that about two weeks after leaving there, he noticed a hard lump underneath the skin on the border of the right lower jaw. About a week later, it had been cut while he was shaving; there had been a serous discharge and the formation of a crust. Under local antiseptic applications, the lesion had slowly enlarged to its present size.

There was a firm swelling, about 1.5 cm. in diameter, with a superficially ulcerated, undermined border and a mass of granulation tissue protruding from the center. Microscopic slides showed numerous large phagocytes containing typical Leishmaniae. A diagnosis of espundia was made.

Inside the left arm, just above the elbow, was a hard, rather elastic, nodular tumor, 2 cm. in diameter, adherent to the overlying skin. This developed as a nodule with a central pore, which had been treated in the native manner by inserting tobacco in the pore. The larvae had apparently become encysted.

DISCUSSION

DR. ELIAS ABRAMOWITZ: About two years ago Dr. Rosen and I saw a woman who had just returned from British Guiana with lesions on her back that looked like ordinary furuncles. A large lesion in the interscapular area showed a watery discharge and every now and then a protruding wormlike body. The entire furuncular lesion was excised, and the contents showed a pear-shaped parasite similar to those shown to us tonight.

DR. FRED WISE: The case is interesting from several aspects. To my knowledge, it is the first case of espundia that has been presented in New York. I did not know, however, that it had been definitely established that espundia is due to infection with the Leishman body. I should like to hear more about the etiology of the disease.

DR. J. GARDNER HOPKINS: Regarding Dr. Wise's question, a great many local popular names have been given to this disease. I am not sure that espundia is the common name for it in Honduras, where this was contracted. The oriental boil and the Aleppo button, as well as espundia and various sores that occur on the legs called forest yaws in South and Central America, are all due to infection with Leishmaniae. In cultures, the parasites have been indistinguishable. There are, however, definite differences in the clinical characteristics of the disease as it occurs in Asia and America, and I do not think that parasitologists are convinced that the parasites are identical. That of espundia is transmitted, as a

rule, by a small fly. It is endemic in dogs, which are apparently the reservoirs of the infection. It seems to be frequently found in men working in the woods, where these flies are numerous. The period of incubation is rather long. This patient stated that the sore did not appear until two weeks after he came back to the United States. The organisms are numerous in the scrapings from the lesion.

As to his other disease, myiasis, the patient has had a number of these lesions. He said that it is common among the men working with him. What he came for was a suggestion as to how to keep the larvae off. He said that these nodules occurred in the skin with a sore that exuded the same sort of fluid that Dr. Rosen mentioned. The native treatment was to insert a piece of tobacco or to close up the pore with gum; then the parasite died and was extruded.

DR. MARION B. SULZBERGER: This case has been of extreme interest to us all. I heard Fülleborn of Hamburg state that cultures which he had made from the organisms infecting the skin in different parts of the world were the same. They are related to the species of Leishmania that causes kala-azar. However, the cultures obtained from kala-azar and skin leishmaniosis show a difference. One type of organism is mobile; the other is not.

DR. J. GARDNER HOPKINS: The favorite method of treatment in Smyrna is with carbon dioxide snow. I think Photinos treats such patients with emetine hydrochloride injected around the lesion. As Dr. Sulzberger said, there are two distinct species of Leishmania: L. donovani, found in kala-azar, the systemic disease, and L. tropica, found in the skin disease. The parasites found in the various types of cutaneous leishmaniosis are indistinguishable.

SECONDARY SYPHILIS. Presented by DR. EUGENE TRAUB.

S. W., a woman, aged 24, married, white, made her first visit to the clinic on April 6, 1931, presenting a generalized eruption composed of discrete and confluent, dull red, infiltrated papules and plaquelike lesions. The scalp was covered by thick, hard scale, with definite suppuration in places. Some of the lesions were follicular. The eruption was confined to the scalp, face trunk and upper extremities, and was of two months' duration.

On Oct. 24, 1930, during pregnancy, the patient had a Wassermann reaction of 3 plus. Since October 24, she had received thirteen injections of neoarsphenamine (0.6 Gm.) and thirteen of a suspension of bismuth salicylate oil (containing 10 per cent of bismuth salicylate). On December 19, the Wassermann reaction was 3 plus. On December 26, an apparently normal child was born, but no Wassermann test was done on the child. On February 15, a generalized rash appeared on the mother, which had steadily progressed.

DISCUSSION

DR. ISIDORE ROSEN: I suggest the diagnosis of arsenical dermatitis. The generalized papulosquamous and eczematous eruption exhibited may follow the administration of arsphenamine.

DR. LOUIS CHARGIN: I feel that we are not dealing with secondary syphilis. The eruption resembles pityriasis rubra pilaris. It is true that one sees papular lesions of this type, especially in women, in follicular syphilis, but there is a great tendency in that type of syphilis for grouping, which is absent in this case. I do not know of an arsphenamine dermatitis of this type. To a great extent this eruption resembles psoriasis, and this condition should be considered.

If it is correct that this patient has late secondary syphilis in the third or fourth year, the eruption should certainly not be symmetrical. It should be unilateral and not so extensive, nor should there be so much scaling. There should be a tendency to ulceration. These points are against late syphilis.

In early secondary syphilis it is possible to have such an eruption so generalized. We have a history that four months ago the patient had thirteen injections of neoarsphenamine, and it is unlikely that a recurrence of such a generalized type would ensue so soon after this treatment. I have never seen so marked a psoriasiform type of dermatitis following arsphenamine. I have seen no such eruption following bismuth either, nor following a combination of the two. It may be an unusual type of arsphenamine dermatitis, but I think that the eruption is syphilitic.

DR. BOLESLAW LAPOWSKI: This is a syphilitic eruption — papulonodular. On both scapulae there is an eruption consisting of distinct syphilitic papules — flat and annular in form. That alone would establish the diagnosis of syphilis, with a subsequent drug dermatitis. Psoriasis can be excluded.

DR. LEO SPIEGEL: I believe the case is one of late secondary syphilis, possibly of eight or nine months' duration. The papules under the breast and on the shoulders are large, flat, infiltrated lesions, as Dr. Lapowski stated. The dermatitis is due to the neoarsphenamine or bismuth, probably to both drugs.

DR. CHARLES WOLF: There are a few points that have not been emphasized. The patient presents crusted, oozing lesions on the forehead and scalp. I cannot conceive of an early or late secondary syphilitic eruption causing such lesions. In addition, the patient presents lichenoid follicular papules, which fits very well the cases of lichen planus-like eruption following the administration of arsphenamine. Determination of the arsenic content in the blood and urine might give some information.

DR. EUGENE TRAUB: It is my belief that this patient has an unusual type of syphilitic eruption, that the patient's syphilis dates back probably about nine months, and that the present appearance is partly because it has been modified by the previous arsphenamine and bismuth treatments, and probably also in part by the preceding pregnancy. I agree with Dr. Lapowski and Dr. Spiegel that the large flat, papular lesions with the great amount of infiltration, particularly over the back and about the undersurface of the breasts, could by themselves be taken for nothing else but syphilis. Whether the follicular lesions particularly marked on the arms are due to arsenic rather than to syphilis, only further observation can determine. The facts that the patient has received four injections of arsphenamine and four injections of bismuth (of the total of thirteen of each received) since the appearance of the eruption, and that, according to the physician who has had her under observation, these additional treatments have caused improvement in the appearance of the eruption suggest, of course, that the condition is essentially not a drug eruption.

IDIOPATHIC MACULAR ATROPHY AND LUPUS ERYTHEMATOSUS. Presented by DR. DAVID BLOOM.

A. M., a woman, aged 30, showed on both ears, more marked on the left, scarring, crusting and telangiectasia, which had commenced when she was 10 years of age. In the right temporal region there was a white, somewhat depressed scar, the size of a half dollar, and on the right side of the face a similar nickel-sized scar. The lesions on the ears had been treated years previously with x-rays, and the scars on the cheek had followed treatment with solid carbon dioxide five years previously. On the upper part of the back and on the shoulders were numerous pea-sized, round and oval, elevated, soft tumors, redder than the normal skin. On the left eyebrow and the right side of the chin there were similar lesions, but of harder consistency. The lesions on the neck were flat and showed distinct wrinkling of the skin, and the palpating finger could easily get through, as though through a hole. The duration had been three months. On the chest, over the manubrium sterni, there were erythema and red spots, somewhat elevated or flat, of the size of the described lesions. The lesions on the back, neck and chest had started eight years prior to presentation.

A biopsy performed on tissue from one of the elevated lesions showed atrophy and disruption of the elastic tissue, edema of the corium and some perivascular infiltration.

The patient was seemingly in good health, and gave no history of tuberculosis or syphilis. She had had three healthy children. The result of the Wassermann test was negative.

PITYRIASIS ROSEA (SECOND ATTACK). Presented by DR. SAMUEL FELDMAN.

R. O., a young woman, aged 21, single, born in Russia, first called at the New York Post-Graduate Hospital Clinic on July 27, 1928. She had been treated at that time for pityriasis rosea, which had lasted two and half months. She remained well until the middle of March, 1931. When on April 6, 1931, she presented herself again at the clinic, she showed over the abdomen and chest and also on the upper arms and axillae, numerous rose-red, macular lesions, varying in size from 0.5 to 3 cm. The centers of some of the lesions showed fine scaling. The configurations were oval and round. The patient was presented because of the rareness of second attacks of pityriasis rosea.

SYPHILIS, URTICARIA PIGMENTOSA AND HAIRY TONGUE. Presented by DR. HERMAN GOODMAN.

A man attended the genito-urinary department of the New York Skin and Cancer Hospital for gonorrhea on Oct. 29, 1930. The result of the Wassermann test of the blood at that time was negative. While he was under treatment for gonorrhea, an eruption developed on the shaft of the penis and on the scrotum, February 26. It was composed of shiny, pale, slightly violaceous papules, about the size of a pea. The patient said that the condition had started after the use of a salve which he had applied to the penis. The result of the Wassermann test on March 10, 1931, was 4 plus.

The patient also presented a generalized eruption of four years' duration, composed of brownish, pea-sized macules and papules. Definite wheals could be easily demonstrated by slightly scratching the skin. On the dorsal surface of the tongue was a brownish-black, papillary overgrowth in the formation of long, slender, filiform projections.

PITYRIASIS LICHENOIDES. Presented by DR. HERMAN GOODMAN.

When first seen in the New York Skin and Cancer Hospital Clinic on Jan. 24, 1931, the patient presented numerous reddish-brown papules and pigmented macules, of the size of a split pea, some of which had a very slight scale that could be easily detached with the finger. The area most extensively affected was that of the entire trunk with the exception of the sternal and interscapular regions; the flexor surfaces of the upper extremities were also affected. Less numerous lesions were present on the neck and on the extensor surface of the arms and thighs. The patient complained of slight itching at times. The eruption was of two months' duration.

The biopsy report was pityriasis lichenoides chronica. Since March 21, 1931, the patient had received four generalized treatments with the ultraviolet rays, starting with an exposure of four minutes and increasing to one of five minutes, at a distance of 20 inches (50.8 cm.) at weekly intervals. The original condition could be seen on the flanks, as this area had not received any therapy. There had been considerable improvement.

SYPHILITIC ANEMIA. Presented by DR. A. BENSON CANNON.

J. A., a white man, aged 40, had been admitted to the City Hospital on Feb. 2, 1931, complaining of difficult gait, deafness, dimness of vision and general weakness. About eighteen years prior to presentation, he had had gonorrhea and

soft chancre, for which he had had internal medication at intervals. Eighteen months prior to presentation, his vision had begun to fail, and in January, 1930, he was told that he had iritis. The result of the Wassermann test of the blood was negative. Three months prior to presentation, he first noticed that he could hardly hear. This condition had progressed until he was totally deaf.

The patient was married; his wife was living and well, and had had three children, who were living and healthy.

Examination showed a fairly well nourished man, extremely pale and apparently chronically ill. The teeth were very poor and necrotic. The blood pressure was 90 systolic and 72 diastolic. The heart was normal. The liver and the spleen were not enlarged. The abdomen and the lungs were normal. The pupils were unequal and irregular and reacted sluggishly to light and to accommodation. There was inequality of the deep reflexes, especially of the left knee and ankle jerks. The abdominal and cremasteric reflexes were not obtainable.

An eye specialist made a diagnosis of interstitial keratitis postsynachia and plastic iritis, while the neurologist suggested the diagnosis of neuritis.

The urine was normal. The blood count showed 2,100,000 red blood cells, 35 per cent hemoglobin, 7,500 white blood cells and a normal differential count. The patient was given a transfusion of 500 cc. of blood. Five days later, the blood count was 2,900,000 red blood cells and 35 per cent hemoglobin. After antisyphilitic treatment, the blood count was 3,900,000 red blood cells and 65 per cent hemoglobin. The Wassermann reaction was 3 plus with cholesterol antigen, and 2 plus with alcoholic antigen; the Kahn precipitation test gave a reaction of 2 plus.

RHINOPHYMA. Presented by DR. A. BENSON CANNON.

P. S., a laborer, aged 65, had been previously presented before the section in March, 1931, with a very marked rhinophyma. He was again presented to show the excellent result obtained with electrocoagulation.

PARAPSORIASIS IN PATCHES. Presented by DR. LOUIS CHARGIN.

M. S., a man, aged 40, a houseworker, from the New York Post-Graduate Clinic, stated that about one year prior to presentation he had first noted an eruption as a patch on the upper part of the right arm. In the next few months it appeared on the other parts of the body. The clinical aspect of the disease remained unchanged from the time that it first appeared, in spite of various treatments to which he had been subjected.

On examination, the eruption was most marked on the arms, but there were many less marked areas on the trunk, front and back. Some of the lesions were small and more or less round, but most of them were large plaques, measuring 2 or 3 inches (5 or 7.6 cm.) in diameter. These were round, but many streaked. The lesions were isolated and confluent, resulting in many festoons. The eruption was superficial, sharply outlined, uniform and slightly scaly, the scale being small and closely adherent. The color varied from red to yellow-red. There was little itching.

Numerous examinations for fungi revealed none. A section of tissue suggested the diagnosis of parapsoriasis.

DERMATITIS MEDICAMENTOSA (PHENOLPHTHALEIN). Presented by DR. A. BENSON CANNON.

K., a man, aged 32, had had repeated eruptions of various kinds over a period of years. He had taken many medicines for various purposes. On February 25, the patient had a headache and took one 5 grain tablet of acetylsalicylic acid. The next day he became sick; his hands and face started to swell; chills developed, and there was vomiting for about twenty-four hours. Blisters formed on the chest and back and in the mouth. On March 24, when he first attended the clinic,

his face, trunk and extremities were covered with large, bluish-brown spots. The hands and lips were cyanotic. The nails had transverse dystrophic lines.

The results of physical examination were negative. The result of a Wassermann test of the blood was negative. The blood chemistry was approximately normal. The urine showed a slight amount of sugar.

On March 25, the patient received acetylsalicylic acid by mouth and was tested intradermally and by patch test with 0.5 per cent solution of acetylsalicylic acid. There was no reaction.

On March 27 the patient received 0.03 Gm. of phenobarbital and was tested intradermally with 1 per cent solution of phenobarbital and with a patch test of the same solution. On March 28, the results of these tests were negative.

At 12:15 p. m., the patient received a small dose of phenolphthalein by mouth. He was tested with a patch test and intradermally with 1 per cent solution of phenolphthalein simultaneously. Returning home, the patient noticed that he had commenced to itch. About 3 p. m., he noticed that the site of the intradermal test with phenolphthalein had become red "like a mosquito bite" and then that the entire body had become red. An urticarial rash broke out over the body. The temperature rose to 100 F. The patient vomited about fifteen times that night. The next morning all his body, including the face, hands and feet, became extremely edematous. On March 31 and April 2, he was given two intravenous injections of sodium thiosulphate. The edema subsided, and only bluish spots were left over the body and extremities. Blisters formed on the right hand. On April 6, the patient presented a generalized exfoliation, mostly of the hands. At 4 p. m. that day he received 0.03 Gm. of phenolphthalein by mouth, and at 7 o'clock on the evening prior to presentation he broke out again with the rash.

PITTSBURGH DERMATOLOGICAL SOCIETY

JAMES H. FISCUS, M.D., *Secretary*

April 16 and May 21, 1931

LOUIS WILLARD, M.D., *President, in the Chair*

TULAREMIA. Presented by DR. W. H. GUY and DR. F. M. JACOB.

A colored man, aged 46, presented a series of nodules extending from the right wrist to the axilla. At the elbow an open wound marked the site of an incised fluctuating mass. While working in a market on Nov. 5, 1930, he cut the last phalanx of the middle finger of the right hand on a bottle, and the next day he cleaned rabbits. One week later he became acutely ill with chills, fever and prostration, being treated by his family physician for blood poisoning over a period of five weeks. He appeared at the university clinic at the end of the ninth week, at which time the temperature was 99.2 F. A large soft mass at the bend of the elbow was drained, and a small nodule was removed, which showed a histologic picture and areas of necrosis similar to those of acute tuberculosis. Serum forwarded to Dr. McCoy agglutinated Bacillus tularense in a dilution of 1:1,280. A row of firm nodules extended from the wrist to the elbow.

DERMATOPHYTOSIS? ARSENICAL DERMATITIS? Presented by DR. H. G. WERTHEIMER.

J. K., a man, aged 44, was seen by us at the Allegheny General Hospital for the first time on Nov. 8, 1929. At that time he presented an eruption confined to the palms and soles. The hypothenar eminences were atrophic, as was the skin on the palms, which was erythematous and covered with hundreds of yellowish papules the size of a pinhead which looked like pustules at first glance.

They contained no pus, but felt like shot under the skin. The eruption on the feet was similar. A diagnosis of dermatophytosis was made, but treatment up until the present time has not changed the picture. Roentgen treatment, together with all sorts of ointments, has been given. A diagnosis of arsenical dermatitis was considered, but several urinalyses were negative for arsenic. The patient had been getting two 7½ grain (0.78 Gm.) enteric coated tablets of sodium thiosulphate each day for the past three months without a change in the eruption.

DISCUSSION

DR. G. J. BUSMAN: I saw this patient about two years ago. The lesions were distinctly pustular then, and I used wet dressings. I believe that at that time the scrapings were positive for fungi. The lesions have improved since I last saw him.

DR. EMERSON GILLESPIE: The inflammatory and vesicular lesions suggest epidermophytosis. I could see none of the keratotic lesions to suggest arsenical dermatitis.

DR. A. R. WOODBURNE: When this patient was seen two years ago, the picture was entirely vesiculopustular. There were marked weeping and oozing of pus. At that time wet dressings were applied. My diagnosis is dermatophytosis of the hands and feet.

DR. FRED AMSHELL: When the patient was first seen, a diagnosis of dermatophytosis was made, and arsenical keratosis was considered. The eruption was extremely resistant to all forms of treatment. There was a history of taking "drops" for a long time. I think that we are dealing with a case of dermatophytosis in combination with arsenical keratosis.

DR. H. G. WERTHEIMER: The case was presented because of its suggestion of both arsenical keratoses and dermatophytosis. I always considered it a case of fungus infection because of the constant presence of vesicles and pustules. It was also presented for suggestions as to treatment; roentgen treatment, light therapy and salves do not seem to have much effect on the disease.

CAROTENEMIA. Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

T. F., a man, aged 25, was first seen two weeks ago in the Mercy Hospital with a history of a gastro-intestinal disturbance for a period of some months for which he had been limiting his diet largely to spinach and eggs. He had eaten as much as two cans of spinach per day. He had a yellowish-orange pigmentation over all the hyperkeratotic areas of the body, that is, the palms, the soles, the extensor surfaces of the arms and the legs, the shoulders and the forehead. The diagnosis of carotenemia was made, and the blood was examined chemically (a demonstration was presented). No adequate description of pathologic changes or demonstration of the particular areas of pigmentation had been found in either the German or the American literature. An attempt is being made to demonstrate these, and the results will be reported at the next meeting.

NOTE.—By controlling the diet, with the elimination of carotene-containing vegetables, a 50 per cent reduction of pigmentation had been produced within the past two weeks.

DISCUSSION

DR. STANLEY CRAWFORD: Is diabetes present?

DR. A. R. WOODBURNE: This patient has never shown sugar in the urine; he has a blood sugar of 80 mg. per hundred cubic centimeters, and gave a normal reaction to the sugar tolerance test.

The technic of the carotene test of the blood serum used was the same as that described by Dr. Fred Wise and Dr. F. A. Diasio in the December, 1929, issue of the ARCHIVES, page 862. This test was used in comparative biology and described by Palmer in 1914, in the *Journal of Biological Chemistry*, and was first reported in clinical medicine by Hess and Meyers in *The Journal of the American Medical Association* (**73**:1743 [Dec. 6] 1919).

PSORIASIS GUTTATA. Presented by DR. LESTER HOLLANDER.

V. L., a man, aged 26, presented a fairly well generalized, symmetrical eruption on the face and the upper and lower extremities. The individual lesion was about the size of half a pea and was covered by a scale. The scale was yellowish, as was the lesion on diascopic pressure. The picture was unusual on account of the peculiarly deep red color at the antecubital spaces, the same reddish hue as might be present in corymbose syphilis. It was unusual on account of the lesions, which were closely packed and had a bluish tinge on the hands. The laboratory study had given entirely negative results.

DISCUSSION

DR. G. J. BUSMAN: This case is interesting to me. If the patient gave a positive Wassermann reaction, all would agree that the eruption is secondary syphilis. He had a penile sore, and considering 60 per cent of penile sores in a sexually active male as syphilis, a sore throat, epitrochlear and cervical adenopathy and an infiltrated, red, papular rash all favor the diagnosis of secondary syphilis. A negative Wassermann reaction is even possible. I would give him old arsphenamine for a therapeutic test and make a daily Wassermann test to see if the treatment provokes a positive reaction.

DR. STANLEY CRAWFORD: I consider this a case of lichen planus.

Dr. F. M. JACOB: I agree that the case is one of lichen planus.

DR. W. H. GUY: I agree with the presenter that it is a case of psoriasis.

DR. LESTER HOLLANDER: The lesions in the antecubital spaces had the appearance and color of late secondary syphilitic lesions, but a completely negative serology did not corroborate this diagnosis. Of course, this does not exclude the diagnosis of syphilis. There are lesions on the scalp, which would be most unusual in lichen planus.

GRANULOMA? Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

Mr. A. R., a man, aged 23, was seen in the Mercy Hospital dispensary two weeks ago with a large dirty ulcer involving the dorsal surface of the left hand and a smaller lesion on the dorsal surface of the left wrist. The ulcer had an overhanging ragged border and a dirty necrotic floor. There were some small abscesses about the border, and a yellowish pus drained from the lesion. A diagnosis of tuberculous ulcer was made.

The lesion began as a small tumor on the dorsal surface of the hand; it was incised about six weeks ago, and a small amount of seropurulent material was expressed. The lesion had continued to enlarge up to the present time. The patient gave a history of acute gonorrhea three years ago, but no primary or secondary syphilis was noted. The Wassermann and the Kahn reactions of the blood were 4+. Roentgenograms of the ventral and lateral surfaces of the hand showed rarefaction of the bone with periosteal thickening near the joints. There were no areas of bony destruction. The suggestion that perhaps the rarefaction was due to disuse was made by the roentgenologist. The cultures revealed no Actinomyces, Blastomyces or Sporotrichum. Staphylococcus albus was the only organism grown. Biopsy revealed a microscopic picture clinically of tuberculosis (a section was shown). Antisyphilitic treatment was instituted last week.

DISCUSSION

DR. F. J. JACOB: I consider this a case of tuberculosis of the subcutaneous tissue.

Dr. C. B. NORRIS: I should give antisyphilitic treatment.

DR. LESTER HOLLANDER: I consider it a case of tuberculosis colloquativa and should not give iodides for fear of disseminating the tuberculous process.

SCLERODERMA. Presented by DR. LESTER HOLLANDER.

J. K., a boy, aged 17, presented a symmetrical involvement of the skin and underlying tissues of the upper and lower extremities, extending to the elbows and knees, respectively. The skin over the hands, especially about the wrists, was reddish, seemed swollen and was firmly fixed. Movement of the involved joints was practically fixed.

Roentgen examination showed a decrease of the joint space, with destruction of the articular cartilages. This condition was of seven months' duration. The patient had an eosinophilia of 16 per cent.

DISCUSSION

DR. STANLEY CRAWFORD: I saw no evidence of scleroderma of the skin of the fingers, hands or wrists, and I consider the condition one of arthropathy, possibly due to the inflamed articular surfaces in the joints of the wrists and fingers.

DR. LESTER HOLLANDER: The skin is swollen, red and edematous, and on palpation it feels fixed; this preceded the fixation of the joints.

A CASE FOR DIAGNOSIS (LUPUS MILIARIS DISSEMINATUS?). Presented by DR. H. T. PHILLIPS and DR. W. J. MORGINSON.

A. F., a white girl, aged 16, single, in July, 1930, first noticed about six minute, discrete, closely grouped nodules on the right side of the upper lip. The nodules gradually coalesced, forming a raised, moderately indurated, noninflammatory plaque, 1 by 0.75 cm., which was a dark reddish brown, studded with small, opaque, solid, tapioca-like, apple jelly-colored areas, about 3 mm. in diameter, and was sunken slightly below the surface of the lesion. The lesion remained stationary for two months, and then increased slightly in size. There was no itching or subjective symptoms.

In November, two similar lesions appeared on the right side of the lower lip; one measured 2 by 1 cm., and the other 1 by 0.5 cm. There were a few scattered, smaller, peripheral lesions. These corresponded in description to the area on the upper lip. They had not changed in appearance since the patient was first seen by us in October.

The area on the upper lip received 2⅓ unfiltered skin units of the roentgen rays in subintensive doses, and the area on the lower lip, 1⅓ skin units of the roentgen rays from October, 1930, to January, 1931. Solution of potassium arsenite was prescribed continuously from December, 1930. The Wassermann reaction was negative. Electrodesiccation of the area on the upper lip resulted in a sunken dark scar. No biopsy was attempted owing to the location of the lesions. Tuberculin tests were not performed.

General physical examination gave negative results. There was no history of tuberculosis in the family.

DISCUSSION

DR. LESTER HOLLANDER: I consider this case an example of miliary lupoid of Boeck. The eruption is composed of small, fine, yellowish nodules that form small atrophic lesions.

A CASE FOR DIAGNOSIS. Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

Mrs. Y. P., aged 22, the mother of several healthy children, presented a marked configurated atrophy with slight wrinkling of the skin of the chest, the upper area of the breasts and the upper part of the arms. On the dorsal surface of the neck, there were linear, hyperpigmented and hyperkeratotic ridges. The ridges stopped abruptly on the lateral surface of the neck.

The condition of the neck had been present since childhood, and had been marked by periods of acute pruritus, erythema and burning. The condition of the chest, arms and breasts had developed gradually over a period of three months.

We saw the patient one year ago, at which time the condition of the neck was virtually the same as it is now. At that time, a mild tar ointment was used without benefit.

DISCUSSION

DR. LESTER HOLLANDER: I consider this a case of lupus erythematosus.

DR. A. R. WOODBURNE: The lesion on the dorsal surface of the neck was identical with that in a case described as one of acanthosis nigricans. The macular lesion over the chest shows no infiltration to substantiate the diagnosis of lichenoid scleroderma. I believe that the condition is macular atrophy.

LICHEN CHRONICUS SIMPLEX? Presented by DR. LESTER HOLLANDER.

P. J., a Negress, aged 18, presented an irregularly scattered, rounded and elongated eruption about the thighs and the abdomen of six months' duration. The lesions were composed of small papules that were covered with a scale; a fine infiltration and pigmentation were present. The patient seemed to have an ichthyotic skin and keratosis pilaris of the lower extremities.

DISCUSSION

DR. B. A. GOLDMANN: I agree with the diagnosis of lichen chronicus simplex.

DR. STANLEY CRAWFORD: The condition is papular syphilis.

DR. LESTER HOLLANDER: I shall give this patient antisyphilitic treatment and report the results at the next meeting.

MULTIPLE SARCOMATOSIS. Presented by DR. CLAUDE B. NORRIS.

H. H., an American, aged 50, married, was presented at the January meeting as having sarcoma as well as tertiary syphilis. During the discussion at that time, it was thought by many that the lesions were due to one of the halogens. Since then a biopsy has been made and the diagnosis of multiple sarcomatosis confirmed.

DISCUSSION

DR. F. M. JACOB: I saw the section, and it shows definitely a round cell tumor. The rapid growth is marked.

DERMATITIS HERPETIFORMIS? MYCOSIS FUNGOIDES? Presented by DR. LESTER HOLLANDER.

R. G., aged 60, presented a generalized eruption of multiform character. The primary lesion appeared to be a scratched off vesicle, which showed definite grouping, especially over the back. Pigmentation and superficial scarring were noted. There were three areas that were important in the consideration of the cutaneous picture. There were elliptic, infiltrated lesions on the torso, the nape of the neck and the left ear, covered by an adherent, brownish-red scale, which were most suggestive of the indurated plaque-like lesions of the second stage of mycosis fungoides.

DISCUSSION

DR. EMERSON GILLESPIE: Dermatitis herpetiformis seem to me to be the correct diagnosis. The presence of isolated lesions simulating such a disease as mycosis fungoides is not unusual. These lesions were herpetiform and in patches, with pruritus. Pruritus, of course, may occur in any such type of dermatosis.

DR. G. J. BUSMAN: I wish to suggest the possibility of parapsoriasis. There are some psoriasis-like lesions over the elbows and the outer aspects of the arms. The duration and superficial character of the lesions, the patient's mental state, etc., could well fit in with that diagnosis.

DR. LESTER HOLLANDER: I showed this patient on account of the peculiar patchy eruption, with the query: Is mycosis fungoides going to develop?

LICHENOID PAPULAR SECONDARY SYPHILIS. Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

L. B., a colored woman, aged 38, married, was first seen on April 29, 1931, with a generalized, shiny, firm, papular eruption, most marked over the shoulders, the dorsal surface of the neck, the forehead, the upper part of the arms, the legs and the back. There was a general adenopathy, but no lesions of the mucous membranes could be found. There were no associated subjective symptoms.

A Wassermann test of the blood made at this time gave a 4 + reaction to all antigens. The question arose as to whether we were dealing with a lichenoid, papular, secondary syphilis or with an extensive lichen planus in a colored woman. Because of the distribution of the lesions and the absence of pruritus, we felt that the condition was a papular, secondary syphilis. A specimen was taken for biopsy to rule out absolutely the diagnosis of lichen planus. Biopsy showed in the papule, which was quite vascular, a circumscribed collection of lymphocytes, fibroblasts and plasma cells. About the periphery of this area were seen occasional small capillaries and arterioles around which was a definite marked lymphocytic infiltration. A specimen taken for biopsy was shown.

DISCUSSION

DR. LESTER HOLLANDER: This is an excellent example of a small lenticular syphilitic eruption well classed as lichen syphiliticus, especially on account of the distribution, involving the neck and the face.

DR. A. R. WOODBURNE: Microscopically, the granulomatous patch in the subpapillary layer is surrounded by small arterioles, each of which has a heavy mantle of lymphocytes. We feel that this picture thoroughly rules out lichen planus and fairly conclusively establishes the diagnosis of lichen syphiliticus.

MYCOSIS FUNGOIDES. Presented by DR. H. G. WERTHEIMER.

A. V., a woman, aged 62, was seen for the first time on May 19, 1931, with an eruption of four years' duration. On the face, the extremities and practically the entire surface of the skin was a brownish-red eruption consisting of plaques of various shapes and sizes, some of which were covered with crusts and scales. A few were mushroom-like. Most of the lesions were markedly infiltrated and showed signs of excoriation. The eruption was very pruritic. Interspersed here and there between the lesions were islands of clear skin.

DISCUSSION

DR. W. H. GUY: I agree with the diagnosis as presented, but I think that the blood should be examined and a biopsy made to rule out leukemia.

DR. LESTER HOLLANDER: Before accepting the diagnosis of mycosis fungoides, a drug eruption must be excluded. The patient has taken a considerable amount of "blood medicine."

DR. H. G. WERTHEIMER: The patient was first seen a few days ago, and that is the reason the report is not complete. I believe it to be a typical case as presented. As for an eruption caused by iodide, the picture is certainly not suggestive of it, in spite of the fact that the patient has taken the drug. I shall be pleased to give a more complete report later.

LUPUS VULGARIS OF THE NOSE: TUBERCULOUS DACTYLITIS. Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

J. L., a colored woman, aged 23, married, had a condition involving the tip of the nose which consisted of bluish-red, pea-sized papules, singly and in groups. Around the nares they gave a rather verrucous appearance. Apple-jelly nodules could be demonstrated on diascopic pressure. The terminal phalanges of the fingers of both hands had distorted nodular prominences, which were bony on palpation.

The Wassermann test and urinalysis gave negative results. Roentgenograms of the hands showed a moth-eaten appearance of the terminal phalanges of all fingers. There was no new bony formation, but there was considerable swelling of the soft tissue about the joints. A specimen taken from the nose for biopsy showed many small caseous tubercles in the subpapillary and reticular layers. A specimen and the roentgenogram of the hands were shown.

DISCUSSION

DR. LESTER HOLLANDER: I was glad to see this patient, who resembles one I presented at the March meeting.

DR. A. R. WOODBURNE: We are in a quandary as to just what to do with this type of case and would appreciate any suggestions as to therapy.

DR. LESTER HOLLANDER: I would advise a trial of the Gerson salt-free diet.

DR. G. J. BUSMAN: This condition is most likely the bovine type of tuberculosis.

PSORIASIS OF THE PALMS. Presented by DR. G. J. BUSMAN and DR. A. R. WOODBURNE.

Mr. W. L., a white man, aged 46, married, was first seen on April 22, 1931, with brightly erythematous plaques of dermatitis involving largely the palmar surface of the hands and the wrists. Over this plaque there was a heavy mica-like scale, and the nails showed the typical dystrophy associated with psoriasis. The only other evidence of the disease were typical large patches over the elbows and the knees.

The patient insisted that this was the first attack of psoriasis he had ever had and that it had been present for only from seven to eight weeks.

DISCUSSION

DR. W. H. GUY: I agree with the diagnosis of the condition on the nails and the elbows and I think that the palmar lesions are also psoriasis. However, I think that a histologic examination is necessary to prove the diagnosis of the condition on the palms.

DR. A. R. WOODBURNE: The condition has involuted so rapidly that the patient does not present a satisfactory picture; however, he shows definitely the effect of therapy with sodium thiosulphate.

ICHTHYOSIS HYSTRIX LINEARIS. Presented by DR. LOUIS WILLARD.

A boy, aged 5 years, presented on many parts of the body and the limbs asymmetric, sharply marginated, linear or irregular, verrucous areas; in places, the areas were arranged in curved parallel streaks.

The disease started when he was about 2 years old, and the mother stated, that some areas had resumed a normal appearance. The boy was breast-fed while his mother had pelvic phlebitis; otherwise, the personal and family histories were irrelevant.

The urine had a specific gravity of 1.030, and had shown a trace of sugar, but two subsequent tests gave negative results. Examination of the blood showed: leukocytes, 11,400; eosinophils, 4 per cent; juvenile neutrophils, 5 per cent; mature neutrophils, 28 per cent; lymphocytes, 42 per cent; mononuclears, 21 per cent. The blood sugar was 80 mg. per hundred cubic centimeters.

UNUSUAL DYSTROPHY OF THE NAILS. Presented by DR. LOUIS WILLARD.

A man, aged 24, a clerk, who had noted a change in his thumb-nail and great toe-nails eighteen months previously, presented a peculiar loss of translucency of his finger-nails and toe-nails; they were a dull greenish gray and abnormally

thick, and the distal halves of some of them were detached. The surface was dull, but smooth, except at the sides, where a few fine, rough lines could be seen. There had been no crumbly growth under the nails. The supra-ungual fold was inflamed, but not tender; there had been no pain or suppuration. His toes showed dermatophytosis, and redness and scaling of the sole were present. Two cultures and two microscopic examinations had given negative results. Two Wassermann reactions of the blood had been negative.

DISCUSSION

DR. LESTER HOLLANDER: I believe that the turbidity and swelling of the nails are due to a systemic disease. I associate these changes with syphilis. Ten years ago I reported the case of a patient with nails with a similar amount of turbidity, but with more pigmentary change. This patient showed a constricted sella turcica and restricted fields of color vision. The patient recovered on treatment with anterior pituitary gland extract. Recently, a patient with nails similar to those of the patient under consideration was studied in my clinic, and after repeated tests we proved the condition to be due to syphilis; the condition cleared up under antisyphilitic treatment.

DR. H. G. WERTHEIMER: I recall having seen a similar case several years ago, at which time I did not know exactly what to call it. The patient had a 4+ Wassermann reaction of the blood.

DR. STANLEY CRAWFORD: The patient continually complains of coldness of his fingers, especially in winter; this has been a familial tendency. I think that the symptoms are the result of an impaired vascular supply to the nail matrix, possibly due to arteriolitis or to a pernio syndrome.

MANHATTAN DERMATOLOGIC SOCIETY

M. B. PAROUNAGIAN, M.D., *Secretary*

April 19, 1931

JEROME KINGSBURY, M.D., *Chairman*

SARCOID. Presented by DR. M. B. PAROUNAGIAN.

G. I., a man, aged 30, from the Bellevue Hospital Clinic, presented at the April, 1930, meeting of this society, had a lesion similar to the present one on the right cheek, the diagnosis of which was unanimously accepted as sarcoid. The lesion had disappeared after two small doses of neoarsphenamine and after painting the lesion with a 5 per cent solution of chrysarobin in chloroform.

The new lesion was of two months' duration; it was dull red, slightly elevated and kidney-shaped, and was located on the left malar prominence. The Wassermann reaction was negative, as before.

DISCUSSION

DR. B. F. OCHS: It is rather interesting to know that once in a while sarcoid will disappear under antisyphilitic treatment.

DR. GEORGE M. MACKEE: This case clinically is sarcoid, but syphilis ought to be ruled out before a definite diagnosis is made.

DR. FRED WISE: I agree with the diagnosis of sarcoid. The reason antisyphilitic treatment is administered is that such treatment sometimes cures patients with sarcoid. Dr. Stokes cured some patients with papulonecrotic tuberculid with arsphenamine. There is no reason why some forms of arsenic should not benefit patients with sarcoid.

DR. ISADORE ROSEN: I suggest the diagnosis of the edematous type of lupus erythematosus, which has a tendency to spontaneous involution.

DR. M. B. PAROUNAGIAN: I saw in a textbook that arsphenamine was recommended for treatment in sarcoid, and therefore I gave neoarsphenamine to the patient. I am confident that this patient does not have syphilis. He improved very rapidly under treatment a year ago.

ATROPHY OF THE SUBCUTANEOUS FAT. Presented by DR. FRED WISE.

C. M., a woman, aged 29, was suffering from congenital syphilis and had received a good deal of antisyphilitic therapy, both intravenous and intragluteal. Two and a half years before presentation the right kidney was removed because of hydronephrosis and complicating pyonephrosis. About the same time she contracted gonorrhea from her husband. The Wassermann reaction was positive, and the Kahn reaction was 4+. Examination of the blood gave the following results: sugar, 0.144 mg. per hundred cubic centimeters; chlorides as sodium chloride, 0.445 mg.; uric acid, 2.5 mg., and urea nitrogen, 9.6 mg. The blood count was as follows: erythrocytes, 3,480,000; leukocytes, 8,200; polymorphonuclears, 49; small lymphocytes, 30; large lymphocytes, 15; mononuclears, 5 and transitionals, 1. The color index was 0.907. The basal metabolism was 26 per cent above normal. Roentgenograms showed soft tissue changes and periarticular atrophy of the upper extremities. The patient presented areas of subcutaneous atrophy of both legs, with some atrophy of the overlying skin. In these areas were subcutaneous nodules over which the skin was slightly reddened.

DISCUSSION

DR. GEORGE M. MACKEE: I think that Dr. Wise has given us an opportunity to see a case of that very rare condition described first by Gilchrist, namely, atrophy of the subcutaneous fat. This disease, as described by Gilchrist, produces not only atrophy and disappearance of subcutaneous fat, but atrophy of the skin. Gilchrist's case also presented nodules that could be felt but not seen. This patient presents cutaneous and subcutaneous atrophy, and also nodules, but the nodules are different from those in Gilchrist's case. In this case the nodules are subcutaneous, but they project above the surface of the skin, are red and not particularly hard. Everything considered, I think that the diagnosis made by Dr. Wise is correct.

I agree with the diagnosis clinically, but I do think that, if possible, Dr. Wise should work the case up as exhaustively as the one published by Gilchrist, for the sake of confirmation of the diagnosis and also as an exhaustive study to compare with Gilchrist's case. In such a rare disease as this, a very careful study should be made. I think that in this case particularly, syphilis should be seriously considered as an etiologic factor. In Gilchrist's case, syphilis was not present, so far as could be determined. There was nothing in the history to suggest syphilis. This patient, I understand, has syphilis. It is possible that vascular changes due to syphilis may lead to atrophy of fat.

DR. ISADORE ROSEN: I think that this patient is suffering from more than the disappearance of the subcutaneous fat. The present clinical appearance is the end-process. We are probably dealing with some type of granuloma, the pathologic changes of which were confined mainly to the blood vessels in the subcutaneous fat.

DR. LUDWIG OULMANN: I agree with Dr. Wise's diagnosis. The patient states that the erythematous and infiltrated lesions have not changed since their onset. If this is so, the case is a matter for observation and histologic examination.

DR. ANDREW GILMOUR: I think there should be a neurologic examination to determine whether there is anything in the nerves that would account for the lesions.

CARCINOMA. Presented by DR. I. ROSEN.

C. V., a woman, aged 64, born in Italy, presented a lesion which had appeared on the gum about eight months previous to presentation, at which time the patient had had a molar tooth removed. Four months later, a lesion on the soft palate appeared, which grew progressively larger. The patient complained of slight pain. There was a large bean-sized lesion, with central, cleancut excavation and rolled, infiltrated border. There was a similar lesion below it. There were no palpable glands.

DISCUSSION

DR. FRANK FRASER: If the growth originated in the mucous membrane, it should be designated carcinoma and not sarcoma.

DR. LUDWIG OULMANN: To my mind, this tumor started in the mucous membrane, involving the underlying bony structure. The lesion is situated on the palate. We know that the lymphatic system of this region would involve the preauricular glands, and any swelling of the maxillary glands would have nothing to do with the primary involvement of the palate.

DR. GEORGE M. MACKEE: This case ought to be discussed from a practical standpoint for the sake of the patient. The clinical diagnosis is certainly carcinoma, but before anything is done, the diagnosis ought to be definitely established, and that can be done by a biopsy with safety. It has been pretty well determined that biopsies do no harm if properly performed. Assuming that the condition is carcinoma, the question arises, What should be done for the patient? Because of her age and poor health, in other words, because of her short expectancy of life, I would hesitate to advise a radical operation. Also, a radical operation would be difficult in this location. It is doubtful if a permanent result can be obtained, which is another reason against a radical operation. I would, therefore, favor treating the patient with electrocoagulation followed by suitably administered roentgen rays or radium at the site of the operation and also to the entire neck. In this way it may be possible to hold the disease in check until she fills out her longevity or dies of some other disease.

SYPHILIS. Presented by DR. B. J. OCHS.

E. S., a well preserved, fairly stout man, aged 32, presented on the anterior surface of the left leg three sharply circumscribed, small ulcers, and on the posterior surface of the left leg about fifteen deep, sharply circumscribed ulcers. These extended from the middle third of the leg to above the ankle.

Eighteen months prior to presentation, the patient applied some liniment for a supposed rheumatic attack. Shortly after, there was an outbreak of vesicles and pustules, which later became gangrenous. These gangrenous areas yielded to treatment, except one large one on the anterior surface of the leg, which formed a linear ulcer about 2½ inches (6. cm.) long. The Wassermann reaction at that time was negative, and the diagnosis was periphlebitis nodularis necroticans. Dr. Satenstein made a microscopic examination and reported the condition to be syphilis. The patient was given a course of intravenous injections of arsphenamine, combined with injections of bismuth and iodides by mouth. Some of the ulcers healed and others became worse. After a short rest, another course of antisyphilitic treatment was administered. The condition remained the same, new lesions appearing in greater number, and the old ones healing. The patient was now receiving a third course of treatment. He was presented because of: (1) the absence of a positive Wassermann test; (2) the chronicity of the ulceration and (3) the appearance of new lesions while he was under treatment.

DISCUSSION

DR. M. B. PAROUNAGIAN: I think that syphilis is the diagnosis, and I also regard the case as one of congenital origin.

DR. FRED WISE: I believe that in this patient, careful investigation and protracted observation are necessary to determine the nature of the lesions, which clinically certainly resemble a syphilitic manifestation.

DR. GEORGE M. MACKEE: The eruption looks like syphilis, because there are numerous, small punched-out ulcers, yet there are many factors against syphilis: one is that the eruption is bilateral; another is that the Wassermann reaction is negative; there is also no history of syphilis, and the therapeutic test gave negative results. Even vigorous antisyphilitic treatment will occasionally fail to cure cutaneous syphilis. It might be well to conduct another therapeutic test and to place more reliance on iodide of potash. In addition to syphilis, it is advisable to investigate for some form of tuberculosis.

DR. B. J. OCHS: When I first saw the patient my diagnosis was periphlebitis nodularis necroticans. A biopsy was done by Dr. Satenstein, and his report was tertiary syphilis; he excluded periphlebitis and also tuberculosis. The patient has been taking iodides for almost one month. In spite of this, I noticed new lesions. I do not think that we can consider ecthyma. I base my diagnosis on syphilis simply because the patient compains of pain at night, and also from the course of the disease and the sharp outlines of the lesions. I am certain of the diagnosis, so I presented the case for discussion.

DERMATITIS VENENATA? Presented by DR. FRED WISE.

H. F., a man, aged 24, born in the United States, entered the Post-Graduate Clinic on April 14, 1931. He was a printer and had been employed in a printing establishment for seven years. He stated that the first eruption began six years prior to presentation and was confined to the face.

The eruption for which he was presented was of two months' duration, and was located on the face, the neck, the external surfaces of the arms and forearms and the inner and upper surfaces of the thighs. The eruption on the face and neck consisted of a highly inflamed and crusted eczema with exudation. On the upper extremities he presented a sharply defined, more diffuse type of eczema, with some scaling and crusting in gyrate formation. There were no actual vesicles present. Within this area were sharply defined clear islands of normal skin. On the lower dorsal region there were a few, scattered, lentil-sized, pigmented spots, such as are often seen after an attack of Duhring's disease. In the middorsal area, over the spine, there was a well defined, yellowish patch about 2 inches (5 cm.) in diameter, showing scaling and follicular hyperkeratosis, evidently the early stage of seborrheic eczema. There were also a few scaly areas on the scalp. Patch tests were being performed with various substances that were suspected.

DISCUSSION

DR. ISADORE ROSEN: I am inclined to agree with the diagnosis of dermatitis venenata. The acute onset, swelling, redness and vesiculation strongly suggest this diagnosis.

DR. LUDVIG OULMANN: I regard this condition as eczema of allergic nature, but I do not think that it is due to the patient's occupation. In such a case his hands would be affected, but they were almost entirely free from the eruption.

DR. M. B. PAROUNAGIAN: I am inclined to think that the diagnosis is eczema of seborrheic origin. As Dr. Oulmann said, if it were due to his occupation his hands and forearms would be apt to be involved.

DR. ANDREW GILMOUR: The involvement was bilateral, and I should call it eczema. His hands were free.

DR. GEORGE M. MACKEE: I suspected this case began as eczema seborrheicum, because the patient has seborrhea of the scalp and what appears to be follicular seborrhea of the trunk.

I suspect that he scratched the lesions, and perhaps irritated them with strong ointments until he caused an acute inflammation with exudation, and then he became sensitive to the exudate, and now he has infectious eczematoid dermatitis. I

do not think that this is Duhring's disease, principally because of the distribution. The eruption may have begun, because of the patient's occupation. as dermatitis venenata, but I doubt it. There is, of course, the possibility that the eruption may be due to retention of a heavy metal, lead for instance, but I adhere to my first interpretation, namely, dermatitis infectiosa eczematoides, secondary to eczema seborrheicum.

ANNULAR SYHILID. Presented by DR. M. B. PAROUNAGIAN.

S. C., a colored man, aged 47, was first seen on April 6, 1931. He gave a history of a genital sore appearing during the latter part of September, 1930. So far as it was possible to ascertain, he had had no treatment for syphilis.

The patient had a number of scattered lesions on different parts of the body, although he was presented for the unusually extensive and very artistic distribution of circinate lesions on most of the face. The lesions were superficial and delicate, and consisted of circles within circles. He also had mucous patches in the mouth and throat. The Wassermann reaction was 4+.

BROOKLYN DERMATOLOGICAL SOCIETY

A. M. PERSKY, M.D., *Secretary*

April 20, 1931

A. WALZER, M.D., *in the Chair*

MULTIPLE BENIGN CYSTIC EPITHELIOMA. Presented by DR. B. B. BERKOWITZ.

G. H., aged 40, married, born in Russia, five months previous to presentation first noted pinhead papules on the chin, which rapidly spread to both cheeks, the nose and the forehead. At that time the only subjective symptom was a burning sensation. The cheeks, chin and forehead showed hard papules ranging in size from that of a pinpoint to that of a pinhead, symmetrically arranged, on an erythematous base. Over the surface of some papules were dilated capillaries.

DISCUSSION

DR. J. SKEER: I think that the condition is cystic adenoma of the vascular type (Pringle).

DR. L. CHARGIN: I am inclined to disagree with the diagnosis as presented. I should like to offer a diagnosis of rosacea type of disseminated miliary lupus. In the location, in the distribution and in the character of the lesion, the picture is of that disease. Pringle's disease might be considered, but against this diagnosis is the age at which it developed; it is unusual to see that number of lesions develop in such a short time. Pringle's disease develops slowly. In this case it is very important to perform a biopsy.

DR. W. H. BEST: I disagree with the diagnosis on two points: the size of the lesion and the rapidity with which the eruption has spread.

DR. A. WALZER: My impression is that the condition is dermatitis venenata resembling rosacea. The entire area disappeared on diascopic pressure. I cannot conceive that lupus of four months' duration would not cause destruction.

DR. E. W. ABRAMOWITZ: I think that miliary lupus is a possibility, and I considered that when I examined the patient. I have seen a few cases of miliary lupus, but I do not recall lesions on the cheek with a border sharply limited to the patches. I do not see necrotic or depressed centers, which one sees in miliary lupus. The patient has not had recurrent outbreaks. I cannot agree with

the diagnosis. I think that the patient has a papular dermatitis on the face, possibly due to the wearing of a fur collar. It is difficult to believe that benign cystic epitheliomas would develop in such profuseness in several months.

DR. B. B. BERKOWITZ: When I first saw this patient, three diagnoses came to my mind: dermatitis venenata, adenoma sebaceum and multiple benign cystic epithelioma. From the history obtained, we discovered that the patient used some face powder, but has ceased using it for the past five months, and I believe that the first condition would have cleared up after the cessation of its use. Therefore, that diagnosis may be ruled out. Regarding fur dermatitis, it was noticed that the fur that the patient wears does not come in contact with the areas affected; on the contrary, the areas that touch the fur are apparently normal. Furthermore, there seem to be no acute symptoms, such as pruritus. As to the diagnosis of multiple benign cystic epithelioma, this condition usually occurs in women about 30 or 40 years of age, but I am not able to speak with authority concerning the rapidity with which it develops. The question raised by Dr. Chargin as to whether the condition is miliary lupus is an important one and will be considered.

ERYTHEMA MULTIFORME. Presented by DR. A. WALZER.

R. M., a housewife, who had two children and was four months' pregnant, gave no history of having taken drugs. An eruption on the arms, hands, legs and feet had been present since six weeks previous to presentation. The lesions began as elevated papules and wheals. They gradually extended peripherally, producing annular and half-ringed lesions, with clear centers. There was a palm-sized patch on the left leg in which the border was flat and purpuric, while the center showed punctate red spots. The mucous membranes showed no lesions.

DISCUSSION

DR. W. H. BEST: My diagnosis is erythema multiforme of the erythema iris type. I accept erythema multiforme as a group of clinical conditions that manifest themselves as an erythematous, papular, vesicular or bullous eruption. The lesions are usually purplish, annular or of circinate configuration, favoring the flexor surfaces.

DR. E. W. ABRAMOWITZ: This patient has erythema multiforme, but not the exudative erythema multiforme of Hebra. An odd feature in this case is the fact that the patient has lesions not only on the backs of the hands, but on the palms. The eruption is evidently toxic.

DR. A. WALZER: This case does not fulfil the typical requirements of erythema multiforme Hebra. The lesions are not full rings, but half ones; this is the closest diagnosis that I can make.

MORPHEA. Presented by DR. L. CHARGIN.

A. B., a woman, aged 20, single, a clerk, first noted the condition about one year previous to presentation. Since then the process had gradually spread. The eruption was limited to the upper inner part of the left arm and consisted of an irregular patch measuring about 3 by 2 inches (7 by 5 cm.). The skin was somewhat boardlike, especially in the white areas that, for the most part, occupied the center of the affected area. The parts that were not white were pigmented, soft and wrinkled. There was slight itching at times.

DISCUSSION

DR. E. W. ABRAMOWITZ: I can only offer a suggestion that I have not tried, but am waiting for an opportunity to try. Unusually good results obtained with injections of turpentine have been reported. It might be interesting to try this treatment on the patient. Baking and massage with small doses of thyroid should also be used.

DR. L. CHARGIN: So far as therapy is concerned, the patient is receiving small doses of thyroid, not because I have faith in this treatment, but because it is so frequently employed, with occasional favorable results. The patient also receives an occasional roentgen treatment. The condition has undoubtedly improved since the patient was first seen.

LICHEN PLANUS LINEARIS. Presented by DR. W. H. BEST.

M. P., a man, aged 39, a clerk, had had an eruption since the early part of January, 1931. The condition started as pinhead-sized reddish lesions under the right breast, which spread in linear fashion around the side. Subsequently, numerous similar linear lesions developed on the right side of the trunk, mostly parallel to the first ones. Slight itching was present, especially in warm weather. Three months after onset, the patient showed numerous linear patches following the intercostal nerves and below the costal margin of the right side of the trunk, a narrow linear patch on the right forearm below the elbow on the ulnar side and a linear patch parallel to and over the spinous process of the right scapula. The patches were confined strictly to the right side of the trunk and forearm; some of them ended abruptly at the median line. The patches varying in width from ⅛ to ½ inches (3.17 to 12.7 mm.) and in length from 1 to 4 inches (2.5 to 10.1 cm.) were made up of numerous confluent and discrete, pinhead-sized, flat, shiny papules having a violaceous hue.

DISCUSSION

DR. J. SKEER: I agree with the diagnosis, but I cannot disregard the suggestion of unilateral linear nevus, except that the lesions are not nevoid.

DR. E. W. ABRAMOWITZ: There is no reason to object to the diagnosis of lichen planus, because the condition has a zoster-like distribution. I have seen a few such cases. Lumbar puncture in the treatment for lichen planus originated with Thibierge, and ever since then some physician has periodically attempted to cure lichen planus in that way. Recently a report on cistern puncture as a treatment for lichen planus was made. Pardo-Castello of Havana has applied medical diathermy to the spine (cervical region) and obtained good results in a small series of cases of this dermatosis. I favor local therapy with the roentgen rays given in fractional doses, with the internal administration of yellow mercurous iodide. I have seen several patients who had recurrences of lichen planus, extending over a period of years. One patient whom I treated twelve years ago shows lesions on the body, although he has received practically all of the usual remedies from a dermatologic colleague.

DR. W. H. BEST: I based the diagnosis on the individual lesions that constitute the patches. They are lichen papules. I cannot accept the diagnosis of nevus unius lateris, because the rash came out quickly, the full complement of lesions appearing in about two or three weeks. As to treatment, the patch on the shoulder and arm received two ¼ skin unit doses of roentgen rays. The lesions on the trunk received intensive doses of ultraviolet rays, sufficient to produce exfoliation. Internally, yellow mercurous iodide, ⅙ grain, four times daily was prescribed. I think that the experience of most physicians is that lichen planus will respond better to this drug than to arsenic. I have used both solution of potassium arsenite and Donovan's solution, but the yellow mercurous iodide seems to be of most value.

MYCOSIS FUNGOIDES? Presented by Dr. A. WALZER.

J. A., a man, aged 45, a chauffeur, gave a history of having had an eruption on the body for fourteen years, which never disappeared completely. In January, 1931, the rash apparently became much more extensive. At presentation the eruption was generalized. The body was covered with an erythematous eruption

of various shades and intensity and showed furfuraceous scaling. Although the eruption involved the entire body, there were intervening areas of apparently normal skin. Some of these normal patches were completely surrounded by erythema with a serpiginous outline. All palpable glands were markedly enlarged. The mucous membranes were not involved.

DISCUSSION

DR. J. SKEER: There were plaques of various shades of red and brown, many of them covered with fine scales. There was furrowing, some of the plaques were infiltrated, and on the face the lesions seemed nodular; most of the glands, especially those in the groin, were markedly enlarged, some to the size of an egg. The itching is not as marked as in mycosis fungoides; however, the patient complains of periods when he has itching. I am inclined toward the diagnosis of leukemia cutis.

DR. W. H. BEST: I think that the mottled, erythematous, brownish, squamous condition of the skin, studded with infiltrated plaques, indicates a diagnosis of mycosis fungoides; judging from the infiltrated lesions on the face and various places on the trunk, the condition is probably past the premycotic stage.

DR. A. WALZER: The peculiar appearance of the rash, the shades of color, the configuration and the normal skin within the erythematous areas are commonly seen in mycosis fungoides. The only factor indicative of leukemia cutis is the enlarged glands, but they are also present in mycosis fungoides.

CLEVELAND DERMATOLOGICAL SOCIETY

H. J. PARKHURST, M.D., *Reporter*

Regular Meeting, April 23, 1931

J. R. DRIVER, M.D., *Presiding*

BISMUTH STOMATITIS. Presented by DR. H. F. DEWOLF.

J. E., a man aged 43, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, had noted a burning pain in his gums on April 17, after his twelfth injection of Bismo Cymol (three injections of 1 cc. and nine of 2 cc., starting on January 13). There was a pronounced bluish discoloration along the margins of the gum, and on the inner aspect of the cheeks, especially on the right side, with some necrosis of the gingival margins and pronounced salivation. The skin showed evidences of healed tertiary lesions on the chest and forearms, these lesions having been active in January. The Wassermann reaction of the blood was strongly positive.

DISCUSSION

DR. C. B. NORRIS: We have recently observed the development of bismuth stomatitis in four patients who had previously received courses of mercury.

RECURRENT SECONDARY SYPHILID; SYPHILITIC NEPHRITIS; SCABIES. Presented by DR. H. F. DEWOLF.

M. M., a Negro, aged 30, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, noticed the appearance of a penile lesion in October, 1930. A dorsal slit was made, and six intravenous and five intramuscular injections were administered. He took no more treatment, and on April 10, 1931, after a period during which he had been drinking "raisin jack," marked edema appeared rather

suddenly, involving the penis, scrotum and lower extremities. There was a pustular eruption on the hands, arms, abdomen, legs and lower half of the buttocks. Between the buttocks there were two semilunar lesions about an inch (2.54 cm.) long, which were slightly ulcerated. The Wassermann reaction of the blood was strongly positive. The spinal fluid was normal in all respects. The dark field observations yielded negative results, but the Fontana stain revealed typical Spirochaeta pallida (Dr. Kline). Acarus scabiei was recovered from the skin. The urine contained a large amount of albumin, erythrocytes, leukocytes and a few granular and hyaline casts. The blood pressure was 205 systolic and 130 diastolic. After the administration of small doses of neoarsphenamine and rest in bed, the edema gradually subsided, and the cutaneous lesions were healing.

DISCUSSION

DR. KARL G. ZWICK: I think the alcohol and the syphilotoxin probably acted together to produce the nephritis. In these cases I feel that neoarsphenamine is valuable, while mercury is too toxic. Small doses of neoarsphenamine give better results.

DR. H. G. MISKJIAN: I should like to object to the common habit of refusing to accept a diagnosis of syphilitic nephritis simply because the condition does not clear up under antisyphilitic treatment. In syphilitic nephritis we must consider the amount of damage that has been done to the kidney. If it is severe, treatment will be of no avail. Cases of syphilitic nephritis fall into three groups: first, cases that respond to antisyphilitic treatment because the patients are treated early; second, those that respond in the beginning, but later become stationary, chronic renal changes resulting in the persistence of albuminuria, and third, those in which treatment is of no avail because uremia develops.

DR. H. N. COLE: This is a classic case of syphilitic nephritis. The patient gives a history of infection about six months ago, with a small amount of treatment, and at present shows the relapse symptoms of syphilitic meningitis. There is pronounced edema of the lower extremities, together with an enormous amount of albumin in the urine without enough casts to explain it. Moreover, the urea nitrogen of the blood is 42 mg. The fact that the albumin is decreasing so rapidly under treatment is a good indication that we are dealing with syphilitic nephritis, and with a nephritis that has not yet done irreparable damage to the kidneys.

A CASE FOR DIAGNOSIS (LYMPHOBLASTOMA?). Presented by DR. H. F. DEWOLF.

G. R., a man aged 76, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, had suffered from a severe generalized pruritus for two years, the condition, having been aggravated by heat, becoming worse during the summer. For the last two months there had been a few small furuncles, but most of them had healed. There were a few crusted papules at the vertex of the scalp. The skin of the entire body, excepting that of the hands and feet, was thickened, shiny and covered with fine scales. It was slightly pigmented, and at times there was slight erythema. There were two or three small furuncles in the left axilla. The cervical lymph nodes were slightly enlarged. The patient was somewhat emaciated. Various local applications had brought no benefit. The erythrocyte count was 4,200,000, and the hemoglobin percentage was 90. The leukocyte count was 14,000, 78 per cent being neutrophils, 4 per cent eosinophils and 18 per cent lymphocytes.

DISCUSSION

DR. ROBERT E. BARNEY: There is marked lymphadenitis, especially in the inguinal regions. Eventually I believe this case will prove to be one of the lymphoblastomas.

DR. H. N. COLE: There are a few nodules in the skin also.

A Case for Diagnosis (Schweninger-Buzzi's Disease?). Presented by Dr. H. F. DeWolf.

Mrs. W., aged 34, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, stated that about five years previously, three months after an abdominal operation, about six coin-sized reddish lesions had appeared on the abdomen, left arm and nucha. These lesions were said to have been soft, and neither elevated nor painful. In about two weeks, without any treatment, these lesions had begun to fade, leaving soft, white, sharply outlined areas of atrophic appearance, which had persisted. Some of these areas showed slight ballooning of the skin, which could be picked up easily and felt like dental rubber. The skin covering the lesions on the neck was more tightly bound down, but was also atrophic.

DISCUSSION

Dr. H. J. Parkhurst: The lesions were suggestive of the end-stage of the condition described by Schweninger and Buzzi.

Dr. H. N. Cole: The condition may be of syphilitic origin, and this possibility must be carefully excluded.

Dr. E. W. Netherton: The patient stated that the early lesions were red nodules, which would fit in well with Dr. Cole's suggestion.

Dr. E. J. Arday: The early lesions disappeared rapidly. Serologic examinations will be made.

A Case for Diagnosis. Presented by Dr. H. F. DeWolf.

L. F., a man aged 20, a clerk, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, stated that since childhood he had had a pruritic eruption which had always been worse during the cold months. In February, 1930, he had been treated for scabies without benefit. Subsequently an eczematoid eruption about the nipples had cleared up under local applications and roentgen irradiation. The general health was good, and there was no familial history of cutaneous diseases. There was a sparse eruption of tiny papules on the trunk and extremities, the back being comparatively exempt. There was some tendency to grouping of the lesions, and pigmentation was present on the abdomen and around the hips. The skin over both shins was considerably thickened. The mucous surfaces presented no abnormality. The differential blood count was within normal limits. Search for Acarus scabiei had been fruitless on two occasions.

DISCUSSION

Dr. E. W. Netherton: The condition over the shins is either hypertrophic lichen planus or neurodermite. The eruption looks like that of scabies. Possibly there is an allergic condition.

Dr. H. J. Parkhurst: In addition to follicular papules and lichenification there is a considerable element of seborrheic dermatitis. There may be an allergic factor also.

Dr. H. N. Cole: There is a definite element of seborrheic dermatitis present.

A Case for Diagnosis (Favus?). Presented by Dr. H. F. DeWolf.

F. P., a boy aged 11, from the City Hospital service of Dr. H. N. Cole and Dr. J. R. Driver, had had an eruption on the scalp since early in 1928. According to the mother, no other member of the family was affected. Over the scalp there were many single and confluent patches of grayish scales and crusts, with yellow cuplike areas from 1 to 2 mm. in diameter. Many spores and air bubbles had been found in the hairs, together with mycelia.

DISCUSSION

DR. J. E. FISHER: Clinically I favor a diagnosis of favus.

DR. R. E. BARNEY: There is little visible scarring, and the disease has lasted over three years.

DR. J. R. DRIVER: The condition has resembled psoriasis and has been treated as such.

DR. C. G. LA ROCCO: The scalp seems to lack the odor found in favus.

DR. J. R. DRIVER: If the crusts are removed, the characteristic mouselike odor is present.

A CASE FOR DIAGNOSIS (KERATOSIS FOLLICULARIS?). Presented by DR. H. N. COLE and DR. J. R. DRIVER.

B. S., a boy aged 16, stated that he had always had an eruption on the face, forearms and neck, especially in hot weather. During the last year the scalp had been red, with scaly areas. The general health was good.

Around the hair border there was a bright red band, slightly scaly, and the same process extended over the entire scalp. There was another band extending entirely around the neck and down over the sternum in a V shape. It was composed of tiny, closely set papules, each with a blackish central spine. The intervening skin was more or less erythematous. Similar lesions were found at the corners of the mouth, about the sides of the nose and around the umbilicus, and a few were found in the axillae. Elsewhere the skin seemed normal.

DISCUSSION

DR. H. J. PARKHURST: The tiny erythematous papules, with their spines, some of which were present on the dorsa of the phalanges, were suggestive of early pityriasis rubra pilaris, but a diagnosis of Darier's disease has to be considered.

DR. E. W. NETHERTON: I felt that the definite keratotic plugs on the neck were more in favor of a diagnosis of Darier's disease. I also observed the lesions on the fingers. It was difficult for me to understand the statement that the eruption had undergone occasional involution.

DR. R. E. BARNEY: When questioned closely, the patient said the eruption had never disappeared entirely.

DR. H. G. MISKJIAN: What is the nature of the erythema on the neck?

DR. H. N. COLE: The patient told me that the eruption had been present since his infancy and had never disappeared completely. The erythema on the neck is a condition often observed in cases of Darier's disease, and the dirty spinelike papules on the neck and around the umbilicus, and a few of those in the axillae, are very characteristic of that condition. There are no other known cases in the patient's family.

BASAL CELL EPITHELIOMA. Presented by DR. EDWARD I. STEINBERG.

Mrs. B., aged 67, had always lived on a farm. There had been a lesion on the radial side of the left wrist for twenty-four years, superficially ulcerated and crusted and gradually enlarging. At the time of presentation it was palm-sized and serpiginous, with a denuded surface and pearly border. There were small areas of granulation and small crusted patches within the lesion. Three units of unfiltered roentgen rays had been administered, without any apparent effect on the lesion The Wassermann reaction was negative.

DISCUSSION

DR. H. N. COLE: I agree with the diagnosis. Radium needles are best suited for the treatment of a slowly growing epithelioma of this type. Electrocoagulation is not advisable.

DR. J. R. DRIVER: In this type of case implantation of radium needles is superior to external irradiation.

THALLIUM POISONING PRODUCED BY A DEPILATORY CREAM (ALOPECIA AND MULTIPLE PERIPHERAL NEURITIS). Presented by DR. E. W. NETHERTON.

A married woman, aged 38, had been observed first two days before the time of her presentation, complaining of pain in the legs, loss of sensation in the arms and legs, abdominal cramps, general weakness, loss of hair, impediment of speech and insomnia. Two months previously she had applied Koremlu depilatory cream to her face because of hypertrichosis. Two weeks after starting to use this cream, she had begun to have pains in the arms and legs and disturbances of sensation. She had then consulted seven physicians, and told them that she was using the cream, but they had assured her that this could not be the cause. Therefore she had continued to use it. The symptoms had then become aggravated, and after a month's use of the cream she had begun to lose hair from her scalp. She had continued the use of the cream until she had come under Dr. Netherton's observation, two days previously.

She had difficulty in walking, due to partial toe drop, and there was bilateral partial paralysis of the lower extremities. There were bilateral paresthesia, diminished deep reflexes, pain over nerve trunks and a bilateral Babinski sign. There was more or less diffuse partial alopecia of the scalp, eyebrows and axillae. The voice was husky, and the patient was mentally depressed.

DISCUSSION

DR. H. F. DEWOLF: This case is interesting to those of us who are using thallium in treatment for ringworm of the scalp. Recently several cases of intoxication due to the use of Koremlu cream have been reported in *The Journal of the American Medical Association*. The thallium content of this cream is 7 per cent. Sabouraud recommended that not more than 1 per cent be used. This cream is being sold for 10 dollars a jar, and it costs only 30 cents to produce it.

DR. E. W. NETHERTON: The intoxication produced by the use of Koremlu cream for the removal of superfluous hair is more dangerous to public health than the radiodermititis produced by the Tricho System. The use of this cream may even prove fatal. This patient had consulted several physicians without being told to stop the use of this cream, and it is therefore obvious that such cases as this one should be brought to the notice of the profession in general. At the Cleveland Clinic, within the past two months, we have observed three cases of thallium poisoning, all of which followed the use of Koremlu cream.

SYRINGOCYSTADENOMA. Presented by DR. R. E. BARNEY.

Miss S. L., aged 19, presented numerous large pinhead-sized to small pea-sized, slightly elevated, firm, pink nodules on the eyelids, which were said to have been present since early childhood. Simular nodules of three years' duration were found on the upper part of the chest. A biopsy on tissue taken from just below the right axilla had revealed changes typical of syringocystadenoma.

A CASE FOR DIAGNOSIS. Presented by DR. R. E. BARNEY.

J. T., a man aged 19, had noticed the appearance and disappearance of "lumps" in the legs and thighs for seven months. There had been no pain, except some discomfort in walking due to lesions in the popliteal spaces. On the inner middle aspect of the left thigh there were three slightly painful, firm, subcutaneous nodules varying in size from that of a large pea to that of a cherry. They tended to be linear in arrangement, suggesting a thrombosed vein, and the overlying skin was dull red. Just below the internal malleoli were groups of painless, dull red,

firm, subcutaneous nodules, varying in size from that of a large pinhead to that of a large pea. The dorsa of the feet were also involved. The hands and feet were purplish and felt cold. Both dorsalis pedis arteries were palpable.

A biopsy had been done on tissue taken from the thigh. Deeply seated in the corium there was a large thrombosed vein with a granulomatous type of infiltrate composed of small lymphocytes, eosinophils, plasma cells and some giant cells. Also there were panarteritis and panphlebitis of the smaller vessels in the vicinity of the large vein.

DISCUSSION

DR. JOHN A. GAMMEL: I saw a similar case about two years ago. After the removal of abscessed teeth, the lesions disappeared.

DR. H. N. COLE: The possibility of syphilis or tuberculosis should be considered in this case.

DR. R. E. BARNEY: The vascular disturbance of the hands and feet, together with the histologic observations, suggests the possibility of early Buerger's disease.

NEW YORK DERMATOLOGICAL SOCIETY

J. FRANK FRASER, M.D., *Secretary*

Regular Meeting, April 28, 1931

JEROME KINGSBURY, M.D., *President*

HYPERTROPHIC LICHEN PLANUS WITH VESICLE FORMATION. Presented by DR. JOHN E. LANE.

E. W. L., a man, aged 37, presented hypertrophic and coalescent patches of lichen planus on the dorsal surface of both hands. A few typical lesions were appearing on various parts of the body. He had a few vesicular lesions, and there was one about the size of a large pea on the back of the left hand. When the patient was first seen on Feb. 13, 1931, the lesions were confined to the hands and the glans.

DISCUSSION

DR. H. H. WHITEHOUSE: I accept the diagnosis. There is no question about the bullous accompaniment, but it has nothing to do with lichen planus. I have seen patches of hypertrophic lichen planus like this clear up with treatment by Unna's ointment: mercuric chloride, 2 grains (0.13 Gm.); phenol, 10 grains (0.65 Gm.); diachylum ointment, 1 ounce (30 cc.). It is generally necessary to increase the mercuric chloride to 4, 6, 8, 10 or 15 grains to the ounce before getting the beneficial effect.

DR. J. E. LANE: The patient has had injections of mercuric salicylarsenate, and roentgen treatment.

DR. FRED WISE: In my opinion, the therapeutic effects of arsenic and mercury are manifested only after the patient has received large dosage, up to the point of tolerance.

DR. FRANK C. COMBES: I think well of injection of mercuric chloride. Dr. Trimble used them frequently with excellent results. It is rather painful, but the results are superior to mercuric salicylarsenate.

DR. GEORGE M. MACKEE: You might try medical diathermy to the cervical and upper dorsal spinal regions, as advocated by Pardo of Cuba. We have tried this method with encouraging results. Later filtered x-rays might be applied to the dorsal and lumbar spinal regions. This case is particularly interesting because of the vesicles. The patient has had one on the penis and now

has several elsewhere. Is the vesiculation a part of the lichen planus? In the few cases that I have seen the vesicles develop on or simultaneously with the papules. In this case the vesicles develop in unaltered skin, and they disappear without leaving papules.

DR. WALTER J. HIGHMAN: This vesiculation is an example of the rare lichen planus pemphigoides. The other lesions are hypertrophic, and hypertrophic lichen planus is very obstinate. I have not seen anything help it.

DR. R. H. RULISON: Several years ago some one told me that Sequiera had cleared up such lesions with lumbar puncture. I tried it on a patient who got well, but he had a bad headache following the puncture. The lichen planus disappeared after several weeks.

DR. H. J. SCHWARTZ: I agree with Dr. Wise about mercury and arsenic being pushed to the limit.

PEMPHIGUS OF THE MOUTH. Presented by DR. JOHN E. LANE.

W. G. G., a man, aged 35, was first seen Nov. 7, 1924, with a history of an eruption of the mouth which had been present for three months and which had started about two weeks after getting some compound solution of cresol in his mouth. He had had Vincent's infection of the mouth, and smears showed the presence of these organisms at that time. In addition to this, there were aphthouslike patches in various parts of the mouth. At that time a diagnosis of Vincent's infection with chronic relapsing aphthous stomatitis was made. The patient was not seen again until April 15, 1931. As presented, the whole of the mucous membrane of the cheeks and lips was covered with superficial ulcerations, apparently the base of superficial bullae which had ruptured. The vermilion border of the lips was covered with unruptured vesicles filled with cloudy fluid, and with crusts. The Wassermann test was negative.

DISCUSSION

DR. W. J. HIGHMAN: I do not see how a diagnosis of pemphigus can be ruled out, but I believe the patient has aphthous stomatitis and also Vincent's angina.

DR. H. H. WHITEHOUSE: The history is not that of pemphigus. I have had no experience with pemphigus remaining for that length of time without more loss in weight. I rather think the condition is a local infection or stomatitis—Vincent's or something similar.

DR. FRED WISE: It is difficult to arrive at a definite diagnosis. The points against the diagnosis of pemphigus are that there is apparently no loss of weight or constitutional disturbance, an absence of cutaneous lesions and, in a measure, the fact that the patient is a gentile.

DR. HOWARD FOX: I agree with Dr. Whitehouse's opinion. The long duration and the lack of loss of weight are strongly against pemphigus.

DR. W. J. HIGHMAN: Referring to Dr. Wise's remark concerning the patient's race and nationality, in this country we happen to see more Jews than persons of other races so affected, but along the entire shore of the Mediterranean the disease is not uncommon in gentiles, and I should think the fact that there are more Jews than gentiles in the United States from Mediterranean countries accounts for our impression.

DR. FRED WISE: I think it is agreed among dermatologists that pemphigus vulgaris is far more prevalent in Jews in this country than in gentiles; the Hebrew race is more susceptible to the disease.

DR. HOWARD FOX: Dr. Wise might add another statement of Dr. Schamberg's. He said that pemphigus in this country is chiefly seen in Jews who were born in Europe.

DR. JOHN E. LANE: Of course, I understand that no one can make a positive diagnosis of pemphigus now. As far as possible, drugs as a cause of the condition have been ruled out. If the condition is erythema multiforme of the mouth, then it is as unusual for it as it is for pemphigus to persist so long without other lesions. The patient will be studied further.

PIGMENTATION OF SCLERAE, EYELIDS AND TEMPLES (MELANIN?). Presented by DR. FRED WISE.

Mrs. M. B., a widow, aged 45, applied for treatment at the skin clinic of the Post-Graduate Hospital on the day of presentation. Her general health appeared to be excellent. She had had two children, both living and in good health.

She presented a brownish-yellow discoloration of the sclera of both eyes, and a light brown discoloration of the upper and lower eyelids and of the skin of the temples. The pigmented areas on the temples were well defined at the lower border, and appeared to shade off gradually toward the scalp.

Too little time had elapsed since the patient's visit to permit a complete urinalysis. A hasty examination, however, revealed the following findings: specific gravity, 1.024; reaction, acid; sugar and albumin, absent; a few red cells and a few leukocytes present. A test for melanin and a test for homogenistic acid (ochronosis?) were to be made.

The patient stated that the pigmentation of the sclerae was present in early childhood, and that the discoloration of the eyelids and of the temple appeared during her first pregnancy twenty years previously. There was no history with reference to the use of drugs, eyedrops or the internal administration of medicine, and also to the use of cosmetics.

DISCUSSION

DR. GEORGE M. MACKEE: The only diagnosis I can offer is melanosis or melanoderma.

DR. JOHN E. LANE: I can add nothing to what Dr. MacKee has said. Dr. Wise has ruled out ochronosis, and I agree with him. I have seen only one case; it is extremely rare.

DR. H. H. WHITEHOUSE: I should think that this case might come under the classification of melanosis. What relation childbirth had to its lighting up and spreading is problematic.

URTICARIA WITH PIGMENTATION. Presented by DR. FRANK C. COMBES.

F. R., a woman, aged 24, born in Austria, presented lesions on both legs and thighs, of one month's duration. They consisted of irregularly shaped, well defined, light brown pigmented macules, varying in size up to 2 cm. in diameter. There were several hemorrhagic wheals that did not disappear on pressure. According to the history, these appeared with itching and disappeared in a few hours, leaving pigmentation.

DISCUSSION

DR. J. J. ELLER: I think that the condition is the acquired type of urticaria pigmentosa.

DR. W. J. HIGHMAN: It is ordinary urticaria with consecutive pigmented spots.

DR. GEORGE M. MACKEE: I hesitate to call it pigmented urticaria. By that term is meant ordinary urticaria followed by marked pigmentation—following it at once, perhaps, but without any apparent reason; but in this case the lesions are hemorrhagic, and the pigment is the result of the hemorrhage, as might occur in any of the inflammatory dermatoses, such as pityriasis rosea, eczema, psoriasis, erythema multiforme, etc.

DR. F. C. COMBES: It is rather difficult for me to classify the case. It does not fall into the group of purpuric urticaria, but it might be considered as urticaria hemorrhagica. I agree that the pigmentation is the result of hemorrhage into the lesions. The history is that the lesions are preceded by itching and appear as well defined large bright red papules, which do not change on pressure. There is a definite hemorrhage into the lesions, but the unusual feature is that the lesions disappear in a short while, leaving a pigmented macule.

A CASE FOR DIAGNOSIS (DERMATITIS VENENATA?). Presented by DR. PAUL E. BECHET.

Mrs. F. C. B., aged 29, stated that she had had an eruption on her face for eight months, from which she had never been free though at times there were remissions. The eruption consisted of pinpoint-sized, bright red, vesicular lesions, so aggregated as to form solid patches, yet in other areas there seemed to be normal skin between them. At a moderate distance the face seemed only flushed, but on close inspection the eruption was easily discernible. There were no subjective symptoms. Most careful questioning failed to elicit the use of any cosmetics other than various cold creams.

Four one-quarter skin units of roentgen rays given once a week had had no effect; neither had bland lotions or salves. The case presented all the symptoms of excessive use of cosmetics, yet the history in that respect was negative.

Therapeutic suggestions were requested.

DISCUSSION

DR. GEORGE M. MACKEE: I see cases of that kind and am always puzzled about the classification. This woman has a very distinct eruption on her face. The first thing to be considered is dermatitis venenata, and tests should be carried out to determine the local etiology. Many of these cases, for various reasons, are often called seborrheic dermatitis if associated with scalp lesions, but they do not respond to local remedies or to roentgen treatment. I sometimes wonder if some of them do not belong to the tuberculosis group. I remember receiving a long time ago a letter from Dr. Ormsby in which he expressed the same opinion.

DR. W. J. HIGHMAN: Dr. MacKee has covered the ground, but a capillary congestion of the skin should be considered with a very fine eruption, vesicular instead of the ordinary rosacea. This has some logic, for even in frank rosacea there are patches like this on the face. I do not think it would be so difficult to find the cause of the eczema if it were undertaken in a comprehensive manner.

DR. H. H. WHITEHOUSE: I agree entirely with Dr. MacKee. The case is in the category of local dermatitis. It makes one think there is a condition at the base of it which may develop later.

DR. FRED WISE: In cases of this type one should search for some external cause, such as hair dyes and perfumes, and should try to eliminate the ordinary cosmetics first; if no results are obtained, search should then be made for more obscure and remote etiologic factors.

DR. P. E. BECHET: I made a diagnosis of dermatitis venenata and have watched the case for eight weeks. The patient never used cosmetics. She is now using plain rice powder and is no better. I am at a loss to know what to do for her.

DR. GEORGE M. MACKEE: One point has not been stressed. I asked the patient about it, and her answer confirmed my impression. It is rather typical of these cases that there are few if any subjective symptoms. We all know that dermatitis venenata on the face is very unpleasant, the subjective symptoms consisting of burning, itching, etc. She is not bothered in that way. She minds only the cosmetic appearance. I have noticed this feature in similar cases.

A CASE FOR DIAGNOSIS (ROSACEA-LIKE TUBERCULID OF LEWANDOWSKY?). Presented by DR. J. J. ELLER.

L. B., a woman, aged 29, a chocolate-dipper, from the skin clinic of the New York Post-Graduate Medical School and Hospital, presented an eruption of three months' duration which was said to have started on the left cheek with a few itchy papules. She was first seen at the clinic in February, 1929, at which time there was over the forehead, nose and chin a diffuse mottled erythema with some scaling and a few scattered papules.

As presented, there were lesions on the forehead, nose, cheeks and neck. On both cheeks were patches of violaceous-red erythema in which were closely set millet seed and pinhead-sized papules. The patches on the cheeks were joined over the bridge of the nose. There was also fine telangiectasia in the patches. Along the borders of the patches were pinhead-sized papules; on the neck were numerous discrete papules, some of which were covered with a serous crust. There was also an occasional pustule. The patient also had pityriasis capitis. She stated that her face burned when she was in a warm room.

Besides local applications, the patient had received eleven roentgen treatments consisting of one-fourth skin unit each between Feb. 17 and April 14, 1931.

Tuberculin tests on April 23, 1931, were negative, as was also the Wassermann test. A biopsy was made on April 23, but report has not as yet been received.

DISCUSSION

DR. HOWARD FOX: I do not see why this case cannot be classed simply as rosacea. Women suffer more often from rosacea of moderate severity than do men. The patient is at the usual age for the condition to develop, and gives a history of gastro-intestinal trouble. I consider this a papulopustular rosacea which would probably yield to internal treatment.

DR. E. F. TRAUB: If I understood correctly, the patient has had eleven roentgen treatments, and she definitely states that the eruption has become progressively worse following each exposure. Furthermore, there is a sharp line of demarcation below both eyelids, marking the site shielded by lead goggles. I have seen mild rosacea not infrequently lighted up in this manner by roentgen rays given in perfectly proper doses, but to a person whose reaction to this form of therapy is not favorable.

DR. R. H. RULISON: I do not think she has rosacea. There is insufficient telangiectasia, and the eruption seems to be basically an erythematous papule and not a dilatation of the blood vessels. I have no diagnosis to offer, but I am not inclined to consider it rosacea.

DR. E. R. MALONEY: I agree with the diagnosis of rosacea but not with the tuberculosis part of the diagnosis. I think it was mentioned that it might be a telangiectasic type of tuberculid, but I do not think it resembles that or acne agminata. There is no depressed scarring such as would be found in tuberculosis of this type after eighteen months. There are no papules with necrosed tops such as are found in this type of tuberculid. I think that the condition is a severe type of rosacea, possibly made worse by treatment and by the application of irritating lotions to the face.

DR. R. S. RULISON: The patient told me that the eruption had entirely disappeared after the application of a salve given at the clinic. She stayed away, and the skin was normal until the lesions recurred two months ago. I asked if during that time there was any redness of the face, and she said there was not.

DR. GEORGE M. MACKEE: The clinical appearance suggests rosacea, but the papular element may signify some member of the tuberculosis group. The fact that the eruption has been stubborn and is spread over the entire face does not exclude rosacea, nor does the absence of pustules. A specimen for biopsy has been

taken, and it may show the so-called rosacea-like tuberculid or perhaps lupus milaris disseminatus. I do not believe the eruption is a tuberculid in the sense of papulonecrotic tuberculid with a nontuberculous histology.

DR. J. J. ELLER: I should like to call attention to the group of pustules on the neck, where the patient has had no treatment.

DR. JOHN E. LANE: I agree with Dr. MacKee that it is a stubborn exaggerated case of rosacea. I do not think the fact that there was not much redness left during the intermissions is against the diagnosis, for frequently this type of rosacea subsides, leaving little redness of the skin.

DR. H. H. WHITEHOUSE: I questioned the patient, as did Dr. Traub, and got the same testimony. The lesion began on the left cheek as a papular rash. If it is rosacea, it is an uncommon type, and the roentgen treatment might well cause the condition; she has lesions on the neck like the ones that disappeared on the left cheek. As Dr. Wise said about some other case, we had better first look for the usual and not the unusual or rare things. Roentgen rays have not been used on the neck, but they would exaggerate the condition with the amount of treatment she has had.

DR. FRED WISE: The eruption was considered to be rosacea for a certain period while the patient was under treatment at the clinic. The appearance of a group of yellowish papules on the front of the neck, at the sternolavicular junction, caused the suspicion that the condition was a papular form of tuberculid.

DR. J. J. ELLER: We first thought that this was a case of rosacea, and later changed our diagnosis to tuberculosis on account of the papular-like appearance of the lesions.

NOTE.—The microscopic slides examined since the presentation showed a definite structure of tuberculosis.

SARCOID OF THE HEAD. Presented by DR. FRANK C. COMBES.

P. L., a woman, aged 27, a native of Porto Rico, presented lesions on her right hand. They were of three months' duration and involved the middle phalanx of the first and second fingers. They consisted of smooth, dusky-red swellings with definite borders and deep infiltration. On palpation they were slightly tender. The Wassermann test of the blood was negative. The Pirquet test was positive. The lesions had shown some improvement after the Pirquet test.

DISCUSSION

DR. FRED WISE: The diagnosis lies between sarcoid and some other form of tuberculosis—a form of sarcoid in which bone involvement has been described. This lesion is external and apparently also involves the periosteum or bone. I would suggest that roentgenograms be made of the affected fingers, as well as the chest.

A CASE FOR DIAGNOSIS (SARCOID?). Presented by DR. FRANK C. COMBES.

M. B., a woman, aged 24, a native of Lithuania, presented two lesions on her cheek to the side and below her left eye. They were of about three months' duration and had not changed perceptibly in size or character. They appeared following an acute periorbital inflammation of unknown etiology of several days' duration. They were both smooth and erythematous, and about 1 cm. in diameter. One was slightly raised and brownish. The other seemed to be composed of small intracutaneous nodules. There was no scaling or subjective symptoms. The Pirquet test was negative.

DR. W. J. HIGHMAN: I think the fact that the Pirquet test is negative would be about as strong a reason to rule out sarcoid or any other form of tuberculosis as one could imagine. The girl has red spots, so far as present conditions are concerned, and that is all that can be said.

DR. H. H. WHITEHOUSE: I do not think we can diagnose the condition today.

DR. PAUL E. BECHET: The possibility of early lupus erythematosus should be considered. Some of the lesions seemed to contain patulous follicular openings.

DR. E. R. MALONEY: It is an early case—only three months—but I think it a form of tuberculosis and that after a while she will have the disseminate facial type of lupus.

DR. GEORGE M. MACKEE: I agree that it is likely to turn out to be lupus erythematosus.

TINEA AMIANTACEA. Presented by DR. J. F. FRASER.

M. S., a girl, aged 13, presented a scaly condition of the scalp and a binding together of the proximal portions of the hairs by asbestos-like scales. The condition was of six months' duration. The scales were very thick, dry and covered the entire scalp. Examinations for fungi gave negative results.

DISCUSSION

DR. JOHN E. LANE: I think there is no question of the diagnosis of tinea amiantacea. The lesions will probably clear up quickly by treatment with oil of cade ointment and daily shampooing. There is usually no loss of hair.

DR. GEORGE M. MACKEE: It is a typical case of tinea amiantacea; it is in patches; there is no infiltration, and when you pluck out a hair, scales adhere to it. My impression is that the disease is not very common. I have seen very little of it, and, while it will clear up under the treatment Dr. Lane mentioned, it will return. The cause is unknown. Apparently there are no fungi.

DR. J. J. ELLER: As to the matter of recurrences, I have a patient 8 years of age, the daughter of a physician, who had a similar eruption which cleared up quickly when treated with soap and water and mercuric ointment; but the father said the condition quickly recurred.

DR. GEORGE M. MACKEE: Some one suggested seborrhea sicca. I do not think it is that; the scales are not powdery and loose like those of seborrhea sicca.

A CASE FOR DIAGNOSIS (LUPUS ERYTHEMATOSUS?). Presented by DR. J. J. ELLER.

N. C., aged 27, called at the skin clinic of the Post-Graduate Hospital on April 25, 1931. There was a patch on the upper part of the chin, beginning at the vermilion border of the lower lip and occupying the upper half of the chin. It was well defined with a serpiginous border. There were adherent scales and one or two areas of atrophy. The eruption was of two years' duration. The patient said that he had not had syphilis. The Wassermann test was negative.

DISCUSSION

DR. FRED WISE: I think that clinically the lesion resembles syphilis, but I would not be pinned down to that diagnosis. I have seen lupus erythematosus look like that, and the scarring is more like that of lupus erythematosus than of syphilis. I favor the diagnosis of lupus erythematosus.

DR. H. H. WHITEHOUSE: I agree with Dr. Wise that it is lupus erythematosus.

DR. R. H. RULISON: I think that it is syphilis.

DR. GEORGE M. MACKEE: The clinical features in this case resemble lupus erythematosus more than syphilis. I cannot differentiate clinically between the two, but I think it is lupus erythematosus because of the atrophy, the color—which is brighter than that usually seen in syphilis—and the fact that the active border is not markedly infiltrated; also, there are adherent scales.

DR. E. R. MALONEY: My impression is that it is lupus erythematosus. Clinically, it resembles tertiary syphilis, but I would not make that diagnosis.

DR. HOWARD FOX: I side with those who do not think it is syphilis, and do so on clinical grounds—not on account of the negative Wassermann test. The raised border was not composed of individual lumpy nodules, so characteristic of the late nodular syphilid. I think lupoid sycosis as well as lupus erythematosus should be considered.

TELANGIECTASIA CAUSED BY GRENZ RAYS. Presented by DR. GEORGE M. MACKEE.

A man, aged 35, for many years had an eruption that was thought to be dermatitis herpetiformis. He had been under observation in the department of dermatology and syphilology of the New York Post-Graduate Medical School and Hospital for four years. Three years before presentation grenz rays had been administered to a number of small areas on several parts of his body. The doses ranged from about a quarter to a full erythema dose—approximately from 100 to 500 roentgen units. No area received more than one treatment. About a year later, telangiectasis developed on several areas that had received erythema doses. The telangiectasis was severe in one area on the right arm, and had endured for two years, during which time there had been no improvement. There was no visible atrophy, hyperpigmentation, depigmentation or any other sequelae.

DISCUSSION

DR. W. J. HIGHMAN: One should see if the telangiectasis fades. I saw a case of Bucky's resulting in a telangiectasia, which disappeared. It may happen in this case. It may be that the grenz rays have a different biologic basis from the ordinary roentgen rays.

DR. E. R. MALONEY: I understood Dr. MacKee to say that the patient was treated for dermatitis herpetiformis by the method of general treatment. That would seem to indicate that the treatment had been given over four areas on the anterior surface and four areas on the back. The only one of these areas that shows telangiectasis is on the back. Unless it can be shown that nothing but grenz ray treatment has been used, and that used locally, I do not think it is fair to assume that the condition is entirely due to the grenz rays. It is necessary to eliminate the possibility of the use of roentgen rays over the affected areas.

DR. FRED WISE: One point ought to be brought out in reference to Bucky's treatment in general, that is the question of the calibration of the machine. Bucky claimed that not all the machines that are being used now are properly calibrated, and the question comes up whether the dosage is the one we thought we were giving. However, that does not in any way argue against the evident effects of overdosage.

DR. J. J. ELLER: It is not unusual to have sequelae of this type. We have about six cases after three years. I have some private patients who received but one erythema dose, and the machine had been calibrated properly.

DR. W. J. HIGHMAN: If too hard a ray is used, the patient is not getting grenz rays. To continue what Dr. Wise said, it is important to be assured that grenz rays are administered. If the borderline is passed and the roentgen field entered, the telangiectasia might be explained.

DR. GEORGE M. MACKEE: The patient has never received treatment with roentgen rays of the usual wavelengths. The apparatus was calibrated with care, both for wavelength and intensity. The tension used in this case was 8 kilovolts.

Grenz rays are being used by dermatologists and other practical physicians, the standardization and calibration being established in most instances by physicists and electrical engineers in the employ of the manufacturers. In order not

to discredit the grenz rays nor to criticize Bucky's contentions unfairly, the calibration should be done and frequently checked by a capable physicist. But in practice this is not done. The grenz rays in this case were used as they are being used in practice, and the patient is presented not for the purpose of discrediting Bucky or the grenz rays but simply to demonstrate that sequelae may follow the use of this radiation as it is being widely employed at present.

I gladly admit that the grenz rays are much safer than are roentgen rays of the usual wavelengths, but I cannot grant that wavelengths of two angstrom units are absolutely safe when administered in erythema doses, and smaller doses do not appear to be very efficacious. The doses now being administered in Germany are enormous—many thousand units. Bucky has obtained good results in port wine marks with doses of several thousand units, but I am afraid to employ such large amounts, especially on the face or over large areas. We have had a number of cases of telangiectasias caused by the grenz ray, but as yet we have noted no other sequelae except for slight atrophy and permanent depigmentation. But I do not hesitate to predict that if a large number of patients are treated with heavy dosage—with 8 kilovolts—we will begin to encounter in ten or twenty years, cases of keratoses and even cancer. I hope that more careful technic or an even lower voltage will preclude this possibility.

Dr. Highman mentioned the biologic effect. Wood and Prime have shown, and I believe that it is now the consensus, that the biologic action of roentgen rays are always the same, regardless of wavelength, so long as exceedingly thin tissue is used and scattered rays are excluded. Grenz rays are roentgen rays of exceedingly long wavelength. Their biologic action is the same as wavelengths ordinarily used for therapeutic purposes, namely, very short wavelengths. What has just been said pertains only to the laboratory. When dealing with animal tissue, the mass effect, or the therapeutic effect, varies with the wavelength because of penetration, scattering, secondary radiation, distance from source of radiation, unequal absorption of different layers of tissue, etc. Grenz rays are absorbed mostly by the epidermis and the papillary body, or by the epidermis alone in the case of marked acanthoses and parakeratosis. But the grenz rays, as are all roentgen rays, are heterogeneous. A small percentage of them are capable of penetrating to a considerable depth, and in the case of heavy repeated doses, or even a single heavy dose when the skin is thin, an amount of the shorter waves may be absorbed by the derma sufficient to effect undesirable and even dangerous sequelae.

I desire to have it distinctly understood that I am an advocate of the grenz rays and I admire Bucky's work, but I advise caution. Dr. Highman stated that the telangiectasia may disappear. That is true. I have seen grenz ray telangiectasia disappear, and I have seen telangiectasia due to ordinary wavelengths disappear, usually leaving pigmented and depigmented areas.

DR. J. J. ELLER: An article has just appeared in which Spithoff of Jena described the treatment in eight cases of lupus vulgaris with grenz rays. He used doses of 22,000 R units, which is a great many times the dosage we use for one erythema dose.

Book Review

HISTOPATHOLOGY OF SKIN DISEASES. By DR. LEE MCCARTHY. Pp. 500, with 250 illustrations, 54 in colors. St. Louis: C. V. Mosby Company, 1931. Price, $25.

There is finally a comprehensive original treatise on the histopathology of skin diseases in English. Men have been promising it or projecting it since the mind remembereth not to the contrary, but it has been left for McCarthy to do it. The invaluable work of Macleod was published in 1903, but that was almost entirely on general pathology of the skin. The monumental work of Unna was translated into English in 1896. However, the first does not cover special pathology, and the second is a work of genius, and that fact itself precludes its being a presentation of accepted general knowledge of pathology. McCarthy does produce, surprising as it may seem, a work on skin diseases unique in English, and one that has been insistently needed for more than a generation.

One takes up the book with eagerness; it is a handsome volume and excites high expectations. It may be said at once that it meets them. It is frankly not a work based on original research, but is a presentation of accepted authoritative views on the subject at the present time, and in this fact—in being a representative presentation and not a personal one—much is added to its value. The subject is presented in five hundred pages and is covered in eleven chapters, which present a classification that, by its logical evolution, lends to clearness and comprehension of the subject by the new student. The work covers all dermatology except the common acute infectious diseases with cutaneous manifestations—from typhoid fever to smallpox—the pathology of which is covered in works devoted to them.

The consideration of the various diseases has been carefully planned, and as a result is briefly and comprehensively developed. There is almost no extraneous matter. There is enough symptomatology to indicate clearly just what the author is considering in his pathologic descriptions, but beyond that the volume is on histopathology. Things like the history of various diseases, which one is so tempted to put in, but which can be gotten so easily in innumerable works, are omitted. Thus the amount of space that may be devoted to the subject proper of the work is made much larger and is adequate.

The pathologic descriptions are sharp and clear, and are always accompanied by the necessary illustrations. Good judgment is shown in the amount of space allotted to the various subjects. For example, syphilis is given forty-eight pages while the next succeeding subject, oriental boil, is given three. But, in the subjects given brief allotment, in only one case has the reviewer found them sketchily or unsatisfactorily handled. There is only one exception; there hardly seems enough space given to mycotic infections. This perhaps may be justified on the ground that these are adequately considered in other available texts, and that this is a book on pathology and not on bacteriology or mycology.

The illustrations for the most part are from hand drawings made by Monsieur A. Bessin of Paris. Relatively few are from photomicrographs, 54 are in color and 196 in black and white. The photomicrographs and free hand drawings are beautifully done. The free hand drawings are a delight, and are the best possible teaching illustrations for the text. The reviewer believes that they would have been made more informative for the immature student by giving fuller descriptions of them in the legends than has been done. There is an extensive bibliography following each chapter.

A word should be said about the work as a book. The paper is peculiarly good for its purpose; it has a sufficiently smooth finish to make the illustrations come out as well as can be done, and yet there is no unpleasant reflection to interfere with its reading qualities. The format, binding, type and everything about the book indicate that nothing has been spared. Such a book represents an enormous expense; the preparation of the illustrations themselves involved no small cost; the reproduction of them at the expense of American illustrations, particularly the reproduction in color, has involved a large sum. No publisher would accept this on his own account. (That is no reflection on the author. Arthur Balfour.

for example, in his reminiscenses, casually mentions that he paid the expenses of publication of his early work on philosophy.) Dr. McCarthy has shown his zeal for his specialty and he has not allowed this formidable difficulty to interfere with the publication of this book. The dedication to Mr. Truxton Beale, well known American diplomat, indicates that he, with generosity and scientific enlightenment, helped carry this burden. We present him our compliments and express our appreciation to him.

Dr. McCarthy, by his scholarship, his courage and his extraordinary determination, has put English-speaking dermatologists under obligation to him. By making the histopathology of all recognized dermatoses readily available, this book will be a great influence for stimulating the general study of the histopathology of skin diseases, and will thus advance dermatology among English-speaking physicians.

Any American or English dermatologist who fails to have this book available will confess by the fact lack of interest in his specialty.

Correspondence

TRYPARSAMIDE THERAPY

Dear Editor:—In connection with the article on "Tryparsamide Therapy" by J. W. Lichtenstein (ARCH. DERMAT. & SYPH. **24**:182 [Aug.] 1931), I should like to present two cases illustrating the dangers of placing too much reliance on tryparsamide as an antisyphilitic agent in certain types of neurosyphilis.

CASE 1.—A man, aged 28, had been found to have a strongly positive Wassermann reaction of the spinal fluid and a first zone colloidal gold curve. He had been treated by means of tryparsamide, bismuth and mercury. During this treatment he noticed dimness of vision, and he was given sodium thiosulphate. Tryparsamide had been administered on the basis of a diagnosis of dementia paralytica for which there was no clinical support. It is probable that the case was one of early syphilitic meningitis that needed intensive antisyphilitic therapy rather than tryparsamide. Examination in our eye clinic showed chorioretinitis. Subsequent treatment by means of typhoid vaccine and ordinary antisyphilitic therapy was given, and no further activity was noted during observation.

CASE 2.—A man, aged 43, had had several mild attacks of motor aphasia that lasted a few hours. He had been treated with tryparsamide and bismuth. While under this treatment, he suddenly became aphasic and had moderate fever and herpes labialis. He showed amnesic aphasia, which lasted for fourteen days. The complement fixation reaction of the blood serum was strongly positive. The spinal fluid showed 4 cells per cubic millimeter, a strongly positive complement fixation reaction and a middle zone colloidal gold curve. The patient was treated by means of malarial and typhoid vaccine along with arsphenamine, bismuth and iodides. He has been perfectly well for two and a half years.

These two cases seem to demonstrate that active progression of a syphilitic process may take place under tryparsamide therapy, and the process apparently may be controlled by routine antisyphilitic treatment. It seems evident that clinical experience substantiates laboratory data that tryparsamide is not a spirillicidal drug and is of value only in resistant neurosyphilis and the preparetic case. I would, therefore, question the advisability of urging the practitioner to use it early in all cases of neurosyphilis.

The use of tryparsamide in our clinic is limited to the resistant and preparetic type of case in which fever treatment is not thought advisable owing to the physical condition of the patient.

S. W. BECKER, M.D., University of Chicago.

Directory of Dermatologic Societies *

NATIONAL DERMATOLOGIC SOCIETIES

American Medical Association, Scientific Assembly, Section on Dermatology and Syphilology

George Miller MacKee, Chairman, 200 W. 59th St., New York.
Francis E. Senear, Secretary, 55 E. Washington St., Chicago.
Place: New Orleans. Time: May 9-13, 1932.

American Dermatological Association

W. H. Mook, President, 3720 Washington Blvd., St. Louis.
W. H. Guy, Secretary, 7026 Jenkins Arcade, Pittsburgh.

British Association of Dermatology and Syphilology (Canadian Branch)

Philip Burnett, President, 2 Simpson St., Montreal.
L. P. Ereaux, Secretary, 1380 Sherbrooke West, Montreal.
Place: Toronto. Time: 1932.

SECTIONAL DERMATOLOGIC SOCIETIES

Baltimore and Washington Dermatological Society

Isaac R. Pels, President, 1010 St. Paul St., Baltimore.
H. Hanford Hopkins, Secretary, Johns Hopkins Hospital, Baltimore.

Four Cities Dermatologic Society

Howard J. Parkhurst, President, 316 Michigan St., Toledo, Ohio.
Robert E. Barney, Secretary, 1021 Prospect Ave., Cleveland.
Place: Cleveland. Time: Dec. 12, 1931.

Iowa and Western Illinois Dermatological Association

J. C. Kessler, President, University Hospital, Iowa City.
J. W. Bailey, Secretary, 10th St. and Grand Ave., Des Moines, Iowa.

New England Dermatological Society

Arthur M. Greenwood, President, 416 Marlboro St., Boston.
William P. Boardman, Secretary, 388 Marlboro St., Boston.
Place: Boston. Time: April 13, 1932.

Southern Medical Association, Section on Dermatology

Clifton Brooks Willmott, Chairman, 608 S. 4th Ave., Louisville, Ky.
Emmet R. Hall, Secretary, 1001 Exchange Bldg., Memphis, Tenn.

STATE DERMATOLOGIC SOCIETIES

Louisiana Dermatological Society

R. A. Oriol, President, 921 Canal St., New Orleans.
M. T. Van Studdiford, Secretary-Treasurer, 150 Baronne St., New Orleans.

Medical Society of the State of New York, Section on Dermatology and Syphilology

Edward R. Maloney, Chairman, 853 7th Ave., New York.
George C. Andrews, Secretary, 115 E. 61st St., New York.

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* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA
SECTION ON DERMATOLOGY

Sigmund S. Greenbaum, President, 1714 Pine St., Philadelphia.
Stanley Crawford, Secretary, 200, 9th St., Pittsburgh.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Cyril K. Valade, Chairman, 1604 Eaton Tower, Detroit.
George H. Belote, Secretary, University Hospital, Ann Arbor.

MINNESOTA DERMATOLOGICAL SOCIETY

C. A. Boreen, President, 920 Medical Arts Bldg., Minneapolis.
D. D. Turnacliff, Secretary, Yeates Bldg., Minneapolis.

OKLAHOMA DERMATOLOGICAL ASSOCIATION

James Stevenson, President, 108 W. 6th St., Tulsa.
W. A. Showman, Secretary, 108 W. 6th St., Tulsa.

LOCAL DERMATOLOGIC SOCIETIES

ATLANTA DERMATOLOGICAL SOCIETY

Francis G. Jones, President, 384 Peachtree St., Atlanta, Ga.
Charles A. Wilkins, Secretary, Medical Arts Bldg., Atlanta, Ga.

[illegible] SOCIETY

Abraham Walzer, President, 217 Clifton St., [illegible]
A. M. Persky, Secretary, 1750 Ocean Pkwy., Brooklyn.

CHICAGO DERMATOLOGICAL SOCIETY

Henry E. Michelson, President, 80 S. 7th St., Minneapolis.
E. A. Oliver, Secretary, 55 E. Washington St., Chicago.

CINCINNATI DERMATOLOGICAL SOCIETY

H. Jerry Lavender, President, 716 Provident Bank Bldg., Cincinnati.
L. E. Valker, Secretary-Treasurer, 916 Provident Bank Bldg., Cincinnati.

CLEVELAND DERMATOLOGICAL SOCIETY

Howard J. Parkhurst, President, 316 Michigan St., Toledo.
Robert E. Barney, Secretary, 1021 Prospect Ave., Cleveland.

DETROIT DERMATOLOGICAL SOCIETY

Claud W. Behn, President, 1546 David Whitney Bldg., Detroit.
William G. Saunders, Secretary-Treasurer, 9203 Grand River Ave., Detroit.
Place: Cleveland. Time: Nov. 19, 1931.

LOS ANGELES DERMATOLOGICAL SOCIETY

Irving R. Bancroft, Chairman, 412 W. 6th St., Los Angeles.
Chris R. Halloran, Secretary, 1052 W. 6th St., Los Angeles.

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CHANGE IN DATE OF MEETING OF FOUR CITIES DERMATOLOGICAL SOCIETY

Since going to press the date of meeting of the Four Cities Dermatological Society has been changed from December 12 to December 10.

Iowa and Western Illinois Dermatological Association

J. C. Kessler, President, University Hospital, Iowa City.
J. W. Bailey, Secretary, 10th St. and Grand Ave., Des Moines, Iowa.

New England Dermatological Society

Arthur M. Greenwood, President, 416 Marlboro St., Boston.
William P. Boardman, Secretary, 388 Marlboro St., Boston.
Place: Boston. Time: April 13, 1932.

Southern Medical Association, Section on Dermatology

Clifton Brooks Willmott, Chairman, 608 S. 4th Ave., Louisville, Ky.
Emmet R. Hall, Secretary, 1001 Exchange Bldg., Memphis, Tenn.

STATE DERMATOLOGIC SOCIETIES

Louisiana Dermatological Society

R. A. Oriol, President, 921 Canal St., New Orleans.
M. T. Van Studdiford, Secretary-Treasurer, 150 Baronne St., New Orleans.

Medical Society of the State of New York, Section on Dermatology and Syphilology

Edward R. Maloney, Chairman, 853 7th Ave., New York.
George C. Andrews, Secretary, 115 E. 61st St., New York.

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* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA
SECTION ON DERMATOLOGY

Sigmund S. Greenbaum, President, 1714 Pine St., Philadelphia.
Stanley Crawford, Secretary, 200, 9th St., Pittsburgh.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Cyril K. Valade, Chairman, 1604 Eaton Tower, Detroit.
George H. Belote, Secretary, University Hospital, Ann Arbor.

MINNESOTA DERMATOLOGICAL SOCIETY

C. A. Boreen, President, 920 Medical Arts Bldg., Minneapolis.
D. D. Turnacliff, Secretary, Yeates Bldg., Minneapolis.

OKLAHOMA DERMATOLOGICAL ASSOCIATION

James Stevenson, President, 108 W. 6th St., Tulsa.
W. A. Showman, Secretary, 108 W. 6th St., Tulsa.

LOCAL DERMATOLOGIC SOCIETIES

ATLANTA DERMATOLOGICAL SOCIETY

Francis G. Jones, President, 384 Peachtree St., Atlanta, Ga.
Charles A. Wilkins, Secretary, Medical Arts Bldg., Atlanta, Ga.

BRONX DERMATOLOGICAL SOCIETY

Henry Silver, Chairman, 290 West End Ave., New York.
Theodore Rosenthal, Secretary-Treasurer, 115 W. 86th St., New York.

BROOKLYN DERMATOLOGICAL SOCIETY

Abraham Walzer, President, 217 Clifton St., Brooklyn.
A. M. Persky, Secretary, 1750 Ocean Pkwy., Brooklyn.

CHICAGO DERMATOLOGICAL SOCIETY

Henry E. Michelson, President, 80 S. 7th St., Minneapolis.
E. A. Oliver, Secretary, 55 E. Washington St., Chicago.

CINCINNATI DERMATOLOGICAL SOCIETY

H. Jerry Lavender, President, 716 Provident Bank Bldg., Cincinnati.
L. E. Valker, Secretary-Treasurer, 916 Provident Bank Bldg., Cincinnati.

CLEVELAND DERMATOLOGICAL SOCIETY

Howard J. Parkhurst, President, 316 Michigan St., Toledo.
Robert E. Barney, Secretary, 1021 Prospect Ave., Cleveland.

DETROIT DERMATOLOGICAL SOCIETY

Claud W. Behn, President, 1546 David Whitney Bldg., Detroit.
William G. Saunders, Secretary-Treasurer, 9203 Grand River Ave., Detroit.
Place: Cleveland. Time: Nov. 19, 1931.

LOS ANGELES DERMATOLOGICAL SOCIETY

Irving R. Bancroft, Chairman, 412 W. 6th St., Los Angeles.
Chris R. Halloran, Secretary, 1052 W. 6th St., Los Angeles.

MANHATTAN DERMATOLOGIC SOCIETY

George Miller MacKee, Chairman, 200 W. 59th St., New York.
Mihran B. Parounagian, Secretary, 126 E. 39th St., New York.

MONTREAL DERMATOLOGICAL SOCIETY

Philip Burnett, President, 2 Simpson St., Montreal.
Lemuel P. Ereaux, Secretary-Treasurer, 1390 Sherbrooke St., West, Montreal.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

Joseph J. Eller, Chairman, 100 W. 59th St., New York.
Leo Spiegel, Secretary, 241 W. 100th St., New York.

NEW YORK DERMATOLOGICAL SOCIETY

John Frank Fraser, President, 100 W. 59th St., New York.
Edward R. Maloney, Secretary, 853 7th Ave., New York.

PHILADELPHIA DERMATOLOGICAL SOCIETY

Carroll S. Wright, President, 1402 Spruce St., Philadelphia.
Jacques P. Guequierre, Secretary, 1930 Chestnut St., Philadelphia.

PITTSBURGH DERMATOLOGICAL SOCIETY

J. H. Fiscus, Chairman, 121 N. Main St., Greensburg, Pa.
Claude B. Norris, Secretary, 244 Lincoln Ave., Youngstown, Ohio.
Place: Pittsburgh. Time: Spring, 1932.

ST. LOUIS DERMATOLOGICAL SOCIETY

Richard S. Weiss, President, 3720 Washington Blvd., St. Louis.
M. F. Engman, Jr., Secretary-Treasurer, 3720 Washington Blvd., St. Louis.

SAN FRANCISCO DERMATOLOGICAL SOCIETY

Howard Morrow, President, 384 Post St., San Francisco.
John M. Graves, Secretary, 909 Hyde St., San Francisco.

News and Comment

SECTION ON DERMATOLOGY AND SYPHILOLOGY

Members of the Section on Dermatology and Syphilology at the New Orleans session, May 9 to 13, 1932, are requested to send applications to the secretary, Dr. Francis E. Senear, 55 East Washington Street, Chicago.

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